YALE
MEDICAL LIBRARY

HISTORICAL
LIBRARY

COLLECTION OF

Alfred P. Klebs

240
44
Book

THE GERM ORIGIN OF TUBERCLE

ILLUSTRATED
FROM THE HISTORY OF PHTHISIS
IN VICTORIA

BY

WILLIAM THOMSON, F.R.C.S.

MELBOURNE: STILLWELL AND CO.
1882

THE GERM ORIGIN OF TUBERCLE

THE GERM ORIGIN OF TUBERCLE

ILLUSTRATED
FROM THE HISTORY OF PHTHISIS
IN VICTORIA

BY

WILLIAM THOMSON, F.R.C.S.

MELBOURNE: STILLWELL AND CO.
1882

GERM ORIGIN OF TUBERCLE.

ALTHOUGH the cause and extent of Phthisis in Victoria have been much argued, yet for lack of exact knowledge of the nature of the disease, and absence of statistical data, neither question could be conclusively settled. But now that science supplies the former want, and the vital statistics of the Colony for the census year 1881 the latter, I propose to review both questions on these premises. In so doing I shall begin with stating the now admitted cause of tubercle, and then go on to consider the extent of the disease in the Colony, as depending on that cause; not sharply defining the two co-related states, but as we go along making them illustrate each other.

In 1870, when I ventured to deny a climatic influence over phthisis, the true nature of the disease was neither known nor surmised. Villemin had just performed his famed inoculating experiments with tubercle, as

Niemeyer revived the inflammatory theory of phthisis which Addison had long before advocated. The medical opinions of the time were by those two works brought into active conflict on the relations that tubercle bore to phthisis, and on the genesis of both; a conflict which led on to the grand debate on tubercle at the London Pathological Society in 1873. Prior by three years to that famous tournament it appeared to me possible to gather together a few data in this newly settled Colony to aid in the preliminary investigation. In this way I began the inquiry into the cause of phthisis, which ended in the clinical inference on the negative effect of climate; an inference now confirmed into an exact scientific aphorism.

In the debate referred to, the ablest pathologists of the day discussed the nature of the morbid product, without arriving at any definite opinion as the outcome of their deliberation. From that hour onwards till 1876 the search for the undiscovered cause of tubercle eluding every effort to find it, either objectively or in imagination, had never been out of mind.

Endless tubercular deposits taken from tubercular bodies, human and bovine, were examined; sputa of every variety were tested; every word written on the subject coming within reach was read; while the behaviour of true phthisis as differing from pseudo-phthisis, or diseases often simulating the real one, was, in this wide field for such observation, clinically watched; until at length the idea all at once struck me that the only way to account for every fact in the morbid anatomy of tubercle, and for every phenomenon in phthisical disease in the living body was to attribute them to the eroding action of a parasitic micro-organism. In this inductive manner was sought out—"traced to its lair"—the parasite creating tubercle and leading on by secondary process to phthisis. The objects shown in specimens and in drawings from specimens prepared by histologists were believed to be the required parasites; and, acting upon that firm belief, the bodies were defined and their biology determined exactly as, and indeed almost in the very words in which, they are now defined after later and fuller observation.

Such then is a brief account of the circumstances antecedent to the train of thought that led up to the publication in July, 1876, of the little brochure entitled *The Histo-Chemistry and Pathogeny of Tubercle*,* a work that can now safely bear comparison with anything which has been since published on the subject. That it was the pioneer work on the germ origin of tubercle is undeniable. In it was first enounced the theory which has been lately adopted by Professor Cohnheim and also by Dr. Robert Koch, amplified by their writings, and verified by their experiments. Without its indication those inquirers might never have entered upon the search. This claim to originality is not put forward in the vanity of priority; it is only made for the value it brings to the work, and to the statements that I am about to make as the results of a self thought out problem. That such is the true character of the pamphlet is shewn by the undoubted testimony of Mr. R. L. J. Ellery,

* *The Histo-Chemistry and Pathogeny of Tubercle.* By William Thomson, F.R.C.S. Melbourne, Stillwell and Knight, 1876.

F.R.S., of the Melbourne Observatory, who, in his Presidential address to the Royal Society of Victoria, October 26th, 1882, alluded to it as :—

A brochure published six years ago (1876) by Mr. William Thomson, of South Yarra, entitled, "Histo-Chemistry and Pathogeny of Tubercle," which I referred to in a former address. In this pamphlet he discusses at length the pathogeny of tubercle, and gives his reasons for concluding it to be a purely germ disease. On page 27 he says, "The idea of micrococci being in any way associated with the process of tuberculosis is a recent one; and the explanation of their mode of operation is, at least as far as I am aware, now for the first time in the history of pathology attempted, with what degree of success remains to be seen." What has now been demonstrated by Koch was undoubtedly indicated in Thomson's pamphlet of 1876, and reiterated at greater length, and with fuller illustrations, in another pamplet in 1879, and afterwards by Cohnheim in his work on the Contagiousness of Tubercle, published in 1880, who says, "We must look forward to the day when the '*tubercle corpuscule*' shall have been discovered in the form of a minute organism."

This is the moderate verdict of a scientist who has been familiar with the idea ever since its inception; and who said, "The "little pamphlet will be a great book some "day;" while the late Dr. Day, of Geelong, referred to its importance as enouncing "a great pathological discovery."

When that work was written, medical science had not found a theory of tuberculosis. And when the writer asked—"How would specific "micrococci operate as the proximate cause "of the equally specific morbid process of "general tuberculosis?" he, answering his own query, then for the first time put to etiology, wrote—"I therefore venture to think "they will probably be found to operate as "the exciting, efficient, or, better termed "proximate cause—the *materies morbi* of "tubercle, somewhat in the following "manner." Then, after minutely explaining the mode of action of the micro-parasite in producing the aggregation of giant cells forming a tubercle, first grey then yellow, he concludes:—"In this mass of epithelial "debris, the minute bodies of the microzymes "are set embalmed, or buried as it were, in "the ruins they produced."

The reader may compare that sentence with the following passage from Dr. Koch:—"It "was in the highest degree impressive to "observe in the centre of the tubercle cell "the minute organism which had created it."

Are not the two accounts identical? In like manner, and in the same words, Dr. Koch also holds the parasite itself to be "the proximate "cause or *materies morbi* of tubercle."

Then I further go on to explain how "the "action thus set up by micrococci would "constitute tuberculosis a true mycosis;" adding, "If the explanation here offered be "found true, it will fully account for the "febrile symptoms occurring on every fresh "swarming or multiple of the parasites;" and it is found true. It verifies "The idea "of micrococci being in any way associated "with the process of tuberculosis is a recent "one; and the explanation of their mode of "operation is, at least, as far as I am aware, "now for the first time in the history of "pathology attempted, with what degree of "success remains to be seen."

Is it too much to add that the success then predicted is now complete?

Even in the minor matters of detail, I observed that "By the advocates of the "contagiousness of tubercle, the germs are "usually said to be spread about by the

"drying-up of the sputa, the resulting "impalpable dust flying about in the air "around the sick person; but it has "never before been explained in what these "infecting germs or particles consisted." The cause of tubercle had been up till that time accounted by pathologists to be "a mysterious something," "some kind of "injurious irritation," a "common unknown "cause." I however preferred to believe that "There is no apparent reason why the "microzymes of tubercle should not be as "specific as those of yeast or of syphilis." But I also remarked, in excuse for the failure of the many attempts by able men to discover the objects of research, that "The existence "of the parasites in single monads, and *never* "*in chains or zooglœa*, may have added to the "difficulty of determining their true nature."

Now, what observes Dr. Baumgarten, in confirming the views of Dr. Koch, upon this point? "His description of the organisms "agrees closely with that of Koch. They "were very rarely united in pairs, and *never* "*massed in the so-called zooglœa form.*"

Is not the language here again the same? Does it not describe the same object?

It is also remarked by Dr. Koch and his commentators that "It may be assumed that "sputum dried on the floor, clothes, &c., "retains its virulence for a long time, and, "inhaled as dust, may set up the disease." Hence the reason why "most cases of "tuberculosis commence in the respiratory "tracts, and the infective material first "develops in the lungs and bronchial glands," because "it is probable that the germs enter "the system by the inspired air, adhering "perhaps to the particles of dust which are "carried along with it;" and also because it "may be assumed that the sputum dried on the "floor, clothes, &c., retains its virulence for a "long time, and, inhaled as dust, may set up "the disease"—precisely as is in the pamphlet explained.

The reader can hardly fail to note that the one narrative is almost word for word with the other, nor help drawing a fair conclusion —agreeing with a scientist in this city— (a German gentleman knowing the genius of

his people), that "Dr. Koch has translated "sentences from the pamphlets into German, "had them re-translated into English, and "added an account of his experiments made "to verify the theory he adopted."

Of that corroboration one other point may be touched, because of its importance to the theory. Referring to the reasons why some persons do not become tubercular on exposure to infection, it is held that one would apply to the problem of infection by the lungs, for "It is probable that the bacilli do not get "into the system, even when inhaled, unless "they can develop in stagnant secretion, or "*unless the loss of epithelium facilitates their* "*ingress.*"

So in my *brochure* it is explained how when microzymes, synonym for bacilli,* enter the lungs in breathing, they could not pass by

* From later observations, Dr. Watson Cheyne affirms that micrococci do not grow into bacilli. They are species of a genus. The point is not settled; but, in any case, cannot affect the parasitic theory of tubercle. Dr. Cheyne has already confirmed Koch's observations.—*Antiseptic Surgery*, 1882, page 247, and *Lancet*, 13th May, 1882.

absorption into the circulation through the pulmonary membrane, nor reach beyond the air sacs; but when there detained they, "*having destroyed the epithelium of the air* "*vesicle*, would as they multiplied enter the "orifices of the lymphatics of the lungs "opening in the stomata lying between the "interstices of the epithelial scales, when the "result would be the beginning of phthisical "irritation."

It could hardly be possible for two accounts to more closely agree, though one was written seven years before the other.

Many more instances of agreement could be given were it needful to multiply proofs to strengthen an already strong case. At Geneva, the other day, Dr. Koch *said* of Pasteur that "his views lacked novelty." But of Dr. Koch, do not I now *prove* that his views lack novelty?

While never dreaming to undervalue the proper work done by Dr. Koch, the writer merely states that in 1876 his pamphlet was sent addressed to very many medical savants and university libraries in Germany—indeed

over the Continent; and amongst others in England to Professor Tyndall, who will probably find it amongst the unconsidered trifles in his library. It was probably unheeded, as being too far in advance of the then state of bacterial etiology.

By the dates of publication we can tell that it certainly was not until after my second pamphlet, that of 1879,* had been likewise sent to them, by Mr. Carl Moërlin, of the Melbourne Observatory, through his friend, Professor Neumayer, of Hamburgh, formerly of Melbourne, that either Professor Cohnheim or Dr. Koch, or yet Professor Tyndall, published a word upon the subject. But although they were so unaccountably reticent and close about a work which had been submitted to them for an opinion, yet a very capable judge in such a question, of his own accord, in the ordinary course of reviewing

---

* *On Phthisis and the Supposed Influence of Climate, being an analysis of statistics of consumption in this part of Australia, with remarks on the cause of the increase of that disease in Melbourne.* By William Thomson, F.R.C.S. Melbourne: Stillwell and Co., 1879.

medical publications, wrote of it to the following effect :—

It gives us pleasure to express our strong approval of the author's views as to the etiology of consumption and its communicability from person to person, especially among individuals crowded together in near proximity in ill-ventilated rooms situated in populous centres, and thus breathing over and over again the same air. The author's view is, that the primary cause of true phthisis is an intra-alveolar deposit of dead epithelium cells robbed of their protoplasm, and thus constituting the well-known giant cells. He considers that the cause of this epithelial death is a germ from an already diseased lung which is inspired by the lung of a patient in a condition of health such as to form a nidus favourable for its reception; and that this germ more easily reaches the apex of the lung, thus accounting for the fact of phthisis being essentially an apex disease. We believe that there is truth in the author's theory, and that it will, if pursued, yield good results. It accounts for the undoubted communicability of consumption under certain circumstances, the comparative immunity of persons engaged in certain trades, and the beneficial results of sea voyages. We respectfully recommend the author to pursue this inquiry into the etiology, pathology, and therapeutics of phthisis.—*The Medical Press and Circular*, March, 1880.

From this notice in a widely read medical journal there can remain no doubt of the public recognition of my work. Moreover, that notice appeared in the very month in which Professor Cohnheim's brochure was published.

Nay, more than that. An esteemed artist, a native of Berlin, living in Melbourne, presumed, on the memory of an old friendship, to write to Professor Cohnheim, asking his opinion of the work, a copy of which accompanied the letter. But instead of replying to his friend, Professor Cohnheim a few months afterwards published at Leipsic, in 1880, his own tractate on the Contagiousness of Consumption, in which occurs this parallel passage:—

> We must look forward to the day when the '*tubercle corpuscle*' shall have been discovered in the form of a minute organism.

The learned Professor might have added to his prophecy:—"As was first enounced "in Mr. Thomson's pamphlets in 1876 and "1879." The frank acknowledgment would not have detracted a whit from, but would rather have enhanced, the merit of his own valuable essay.

It was to more thoroughly work out the problem, which had not been fully grasped by the profession, that I again, in July, 1879, returned to the discussion of the question in

a rejoinder to an elaborate *Report* of a select committee in the Medical Society of Victoria, specially appointed to rebut my statements and refute my theory. The new publication was reviewed at very great length in the Melbourne *Argus* of the 25th July, 1879. The lay review was a recapitulation of the *Medical Report* as it appeared in the *Australian Medical Journal* of December, 1877.

To mark the progress of pathology during the interval between then and now, one need only quote briefly their chief conclusion in opposition to the germ theory, that:—

> It is now recognised that most cases of phthisis represent really the final stage of some acute inflammatory affection, mostly pneumonia.

From this postulate it was further affirmed that since ordinary lung inflammations are relatively few here, therefore ought there to be little phthisis; and by that reasoning in a circle was negatived the parasitic theory of phthisis or co-related tubercle.

But, as I have all along maintained, inflammation is not the initial stage of phthisis. The specific irritant acting as a

foreign body in the lung tissue may set up inflammatory action that may quickly destroy the affected organ, but to check that is not to cure phthisis, nor to alter its course in the least. However, having contended against my data and my pathology, they go on to comment how —

> He argues in favour of a theory that phthisis is really a contagious disease. As a theory, it has not commended itself to English physicians generally. Mr. Thomson may be original in the suggestion that the contagious quality is owing to the presence of a micrococcus. (The Melbourne *Argus*, 25th July, 1879.)

Here, then, was a distinct and emphatic recognition of the originality of the new etiology. Whether the "*suggestion*" was mere guess, or the higher induction it indeed is now found to be beyond all cavil, or whether the idea threw light on what was else profound mystery, the medical reviewers stayed not to inquire. But on comparing their pronouncement of July 25th, 1879, with the account given in the same journal on July 12th, 1882, of Dr. Koch's discovery, it is seen how thoroughly they have come round to concur with my views on the parasitic theory

which they were unwisely misled to contemn. After relating the leading facts adduced by Dr. Koch, they sensibly observe:—

It is evident from this that consumptive people are by no means the safe companions they have hitherto been regarded. The facts we have mentioned should suggest precautions which, up to the present time, have been considered unnecessary. Care should be taken to destroy all organisms in the matter thrown off the lungs of patients by immersion in boiling water or otherwise, before it can dry and be taken up by the atmosphere. We should imagine, moreover, that there must be considerable danger in sleeping with people suffering from phthisis. (The Melbourne *Argus*, 12th July, 1882.)

To assert that the precautions against specific infection have not been hitherto thought necessary, is true enough. And whose fault was it? The contagion theory which had "not commended itself to English "physicians generally" did no more commend itself to Colonial physicians particularly. To think for themselves would be beyond all conservative discipline.

But to say that such precautions have never before been declared to be required, is very far from true, as the columns of their own journal bear witness. For example, in the

work reviewed in it in July, 1879, are these passages :—

> The crowding or huddling young folks together in sleeping-rooms and work-rooms facilitates the process of transferring virus. (Page 76.)
>
> If the respiratory movement is carried on in an atmosphere laden with exuviæ from phthisical lungs, inhalation of their infective material and inoculation therewith will readily take place. (Page 79.)

Again, in the inquiry into the cause of consumption among the blacks at the aboriginal station, Coranderrk, the writer, in his evidence, laid great stress on the fact that the sputa of patients were allowed to lie and dry into dust on the earthen floors, and so propagate contagion. What though his notion was hooted at because forsooth it was not the climatic explanation wanted? It was still the true one, as the then rejecter must now admit. (See *Report*, printed by Parliament, 1881.)

My vigilant reviewers could hardly have overlooked those pregnant passages, read side by side along with others of like import.

Had those medical reviewers who made *The Australian Medical Journal* or the leading

lay journal in the colony alternately their organ, according as they would appeal to the medical profession, or address the general public, of colonial or home readers, ignored my writings all along, I should not now have had any claim upon their attention. But seeing they have keenly combated every word put forward by me on the germ theory of disease, and fought me in phalanx with every weapon of fair or unfair dialectic, contending as if their very life depended on my defeat, their own acts require me now to remind them in the triumph of truth over error, how they strove in vain to hinder the progress of science. They full well know what were the views held by me before them for their guidance long ere germ theories about "death's household worms" became familiar in their mouths as household words. The theory of a *contagium vivum* in phthisis was no vague surmise. The purposed induction was to explain facts otherwise inexplicable; it is now a verified inference. That is all. And so I brought the cause of tubercle within the reach of human knowledge.

Of the practical utility of the theory he confirms Dr. Koch says little, and he is commended by professional critics for his caution, though lay critics are exacting.

But in 1876, I ventured to foretell that:—
"Eventually the above theory might become
"directly clinically instructive by pointing
"towards a rational method of preventing
"phthisis, or cure by agents experimentally
"known to be destructive—germicidal—of the
"organisms. For, while it is but a truism
"to affirm that the healing art never can
"restore lost lung structure, so does it seem
"premature to assert the improbability of
"ever finding means of killing parasitic
"particles in living tissue without at the
"same time destroying its own integrity. In
"this, indeed, might be found the true
"*rationale* of the action of antiseptic
"inhalations in phthisis. It would explain
"how the antiseptic becomes directly curative
"of phthisis by arresting the parasitic cause
"of destruction."

Here, surely, is plainly enough set forth, without any ambiguity, technical term, or

involved meaning, both the nature of the disease and the means of curing it. That was printed several years before the principle enounced came into general use by handy mechanical inventions now in vogue, whose contrivers it directed.

Simple people think the efficacy of these respirators resides in the form of inhaler, and not in the antiseptic vapour. Yet, a tea-cup was the earliest, and is still the best, form of naso-oral respirator. It did work many cures of phthisis before our cutlers would go to work.

But the commonly-inhaled carbolic acid often proved hurtful; it checked the disease, but lowered the patient, its poisonous quality making it occasionally dangerous. Hence, on finding this effect of it I, when referring to Pasteur's discoveries, wrote :—"To arrest the "eroding action of bacteria in the air-cells of "the lungs an antiseptic is required that will "not affect the oxygen of the respired air. "This is our great desideratum that has not "yet been touched. To supply it will excel "all that has ever yet been done."

In boroglyceride I have lately tried to find a better and safer inhalant. It is the most efficient curative agent in surgery or medicine. A saturated solution of it in water used as a spray is a most effective means of applying direct medication to the lungs in phthisis. After many trials of it during the past six months; indeed, ever since I read an account of the new antiseptic in the *The Times*, I have found it to be without a rival. In many cutaneous affections — erysipelas, tetters, eruptions with scabs or incrustations, ringworm, porrigo, &c. &c., in which I first tried it tentatively, it acts "like magic," as a patient cured said. By analogy, I thought the remedy might have equal healing powers internally, and it has them. It will not aid phthisis in the incurable stages; but it will make it one of the most curable in the category of curable affections. It fulfils every indication formerly noted by me in *The Germ Theory of Phthisis*, for a true remedy, which would arrest the lung disease, heal ulcers, and cause to be resorbed effused lymph in or around the air cells, without injury to the

blood or normal tissues, and without impairing the quality of the respired air; neither de-oxidizing nor raising the oxygen to a peroxide of hydrogen, changes alike hurtful in phthisis. These are the positive and negative qualities of the boroglyceride, in which it differs from, and therefore excels, all other inhalants. How it produces the healing effect, whether as an aseptic, an antiseptic, a germicide, or all three in one, need not now be discussed. The mode of action may be considered again. The agent is, moreover, wholesome, pleasant to take, easily applied, and little costly.

I shall now proceed to show the importance of this inquiry by the prevalence of phthisis in Victoria, and its increase among the native-born. The statistics of the number of victims to tubercular disease adduced by Koch seem to have astounded everybody. He observes that "one-seventh of all deaths are caused by "tuberculosis." The proportion they bear here cannot be very many lower, probably not a tenth. But this statistical fact is undoubted—that here in Victoria, in 1880, of all deaths

from all causes that occurred in adults between the ages of 15 and 45 years, *one in three was from that form of tubercular disease known as pulmonary phthisis*, or common consumption. Nay, the ratio is perhaps higher; for, as was recently remarked by one of the oldest and best known medical men in the place, within hearing of Mr. W. H. Archer, the ex-Registrar-General, many people not liking the idea of phthisis being in their family, get medical men to assign other than the real cause in their certificate of death. Surely, this high death-rate, which has often been noted, gives the greatest importance to the discovery of the nature of phthisis for the people of Victoria, particularly as the cause can now be so readily attacked and its ravages entirely prevented.

With a view to bring these facts again under public notice, I, in the third week of March, 1882, once more ventured to publish on *The Germ Theory of Disease Applied to Eradicate Phthisis from Victoria.* As it so happened, that was the very week in which Dr. Robert Koch addressed the Physiological Society of Berlin on the parasitic organism

about whose ravages in Victoria I too was writing! Without being in any way aware of what Dr. Koch was then doing, I was endeavouring to apply practically, for the welfare of my fellow-colonists, the very principles and facts that he dealt with experimentally.

The experiments of Dr. Koch verify the formerly enounced theory which led to them. That theory taught how phthisis is a local infection of the mucous membrane of the aërating portion of the lung by fungoid organisms, naturally tending, if unchecked, to spread until the whole organ be destroyed; that the infecting organism is derived always from a prior case of the same disease; and that the disease is therefore contagious. The idea which was scouted as a paradox is now approved science. We have come to a knowledge of the cause of tubercle. And "The End of our Foundation is the "knowledge of Causes; and the enlarging "the bounds of Human Empire over" the hitherto helplessly uncontrolled phthisis. In so being a contagious disease, phthisis has

often enough been imported into every colony in Australia.

From the false fame the climate enjoys for curing phthisis, and the many phthisical invalids flocking hither allured by hope of recovery, the native-born, white and black, have been freely exposed to contagion. The two facts stand to each other in the relation of cause and effect.

That bringers of contagion derive benefit equal to the harm they inflict is a fond belief with many, though actual cases never have been adduced, here or elsewhere, to prove it. And the best proof that cases cannot be given to bear out the former belief is to be found in the fact that the advocates of change of climate for the cure of phthisis now never name Australia as a place for an alternative trip to, when new-fashioned places such as Davos fail.

After that failure—the sorriest medical *fiasco* of modern times—great English physicians cease sending phthisical invalids hither to suffer hardship along with disease, leaving men of minor note to imitative folly. Or sufferers

take their own way, and come; for laymen slowly throw off a popular medical delusion derived from glowing writings in medical fiction.

At the sad fulfilment of prediction the author does not "*rejoice*," as a Melbourne publicist insinuates. I have neither allowed myself to rejoice nor deplore. I have only set forth a fact in the decline of our adult population, to prove the progress of medical science; just as the same fact from the last census of the population is used by the same journalist to show, through unwise laws, a decline in the science of politics. In using their argument our political economists would not care to be set down as diabolic, for their probity.

These remarks are the more called for since the former error of the Victorian Medical Society has lately been revived in a new and more pernicious form. The London Institute of Actuaries re-opens the discussion of a subject which the more thoughtful colonists, including the better Actuaries, considered settled. There I too would have left the question, were I not called upon by name either to tacitly admit

that I, as the leader of opinion, was wrong, or else to vindicate my position against a host of fresh assailants. I prefer the latter as the more agreeable exercise. A defence will also, I trust, prove the more commendable course to the general community, from the important truths that will on their behalf be elicited in the recreation.

In a lengthy debate at the London Institute of Actuaries *On the Rates of Mortality of Victoria*, Victorians will find matter for reflection, as a people provident in life assurance, if of little forethought in avoiding the cause of what their Actuaries know to be their most fatal malady. The eloquent orator on the occasion wound up with a peroration upon the defects he found in my writings upon the statistics of phthisis in Victoria, and is reported to have spoken to the following effect :—

With regard to phthisis, I may make one remark on a publication issued in Melbourne by Mr. Thomson, who makes some exceedingly strong deductions from the figures, and states, in fact, that consumption is not only as heavy as I have stated here, but that it is on the increase. I think, if I may be allowed to say so, after an examination of his book, he falls into a mistake by too strongly dividing the consumption in the country

districts from the consumption in Melbourne. He forgets that almost all the cases occur in Melbourne, probably not only from the reason that they originally lived there, but because sick people naturally go where hospital and medical aid can be most readily gained. But for that reason it is rather hard to say that the consumption of the whole of Victoria shows such a large increase. As a person very happily put it, you might as reasonably say the mortality in a bedroom is much larger than in a sitting-room, where it is not the difference of climate of the two rooms, but because most people go to their bedrooms when attacked by sickness.*

It is hard to know whether to begin with the illogical argument or its unclinical illustration. On neither did Mr. Thomson "mistake" or "forget," as the critic ought to be aware, if he really read *his* book, and does not discuss it second-hand, as critics mostly do. This is, indeed, probably the true explanation of Mr. A. F. Burridge's extraordinary statement; for there was present as his prompter an eminent Actuary from Melbourne, who, in joining in the debate, evinced a marvellous ignorance, altogether Victorian, on a subject with which he, as an active Melbourne Actuary, might otherwise be presumably familiar. At all events,

---

* *The Insurance Record*, London, 14th April, 1882.

in no other way can one so charitably explain why Mr. Burridge made the most inaccurate assertion that almost all the deaths from phthisis in Victoria occur in Melbourne. That I the writer thus criticised did not unwisely so divide the cases into town and country is proved by the table below, reproduced from a former work, and brought down to date. It shows that nearly one-half the deaths from phthisis occur in the country, the other half in Melbourne and suburbs, an area twenty miles in diameter, and containing over a third of the whole population of the Colony.

Town against Country.

Year.	PHTHISIS.		
	Total.	Town.	Country.
1874	1,011	531	480
1875	1,027	525	505
1876	1,010	555	455
1877	1,088	570	511
1878	1,124	580	544
1879	1,058	577	483
1880	1,175	666	509
1881	1,199	647	552
Total	8,692	4,651	4,039

Obviously, therefore, all the deaths do not occur in Melbourne. The overgrown city has its own phthisis, and is jealous of receiving into its hospitals phthisical strangers from the country. From 1871 to 1881 there were 2928 phthisis cases treated in the Melbourne Hospital, with 1286 deaths, or 44 per cent.; and they were almost all town cases. Besides, all the interior cities and towns—Sandhurst, Ballarat, Geelong, Beechworth—have every one a well-appointed hospital, where phthisical invalids in the several districts go more readily than come on to the metropolitan. Nay, more probably by far, many young people come from the country in prime health to Melbourne, to go into domestic service, work-rooms and factories, catch contagion, and return home in sickness, perhaps to die, as I have known several. In discussing the question, I endeavoured to carefully ascertain where the individual cases came from, as far as that was possible, and the results of the long, laborious, and to me costly research are faithfully detailed in my work. *(On Phthisis*, 1870, p. 20.*)*

On the increase of phthisis, the latest Victorian *Year-Book* affirms that the death-rate from this complaint has of late years been increasing. That is, in all Victoria—not any one large centre of population where invalids congregate. In 1871 there were 841 deaths, or 11·41 per 10,000 persons living; and in 1881 these now rise to 1,199 deaths, or 13·80 ratio. This 1,199 is the largest number recorded in any one year. That rate is not quite so high as the English, but it is in a sparser population, with a *far larger proportion of young children*. For the latter reason, there are fewer grey hairs in Victoria. Both effects are *climacteric*, not climatic.

But the most important point of all, which Mr. Burridge overlooked, was started by the Melbourne Actuary referred to, who took part in the debate. He, Mr. T. Jaques Martin, "should like to know the number of native-"born exposed to observation, with the result "of consumption, according to the table which "our valuable statist, Mr. Hayter, has, I "presume, provided in view of the circum-"stances I have referred to." Now Mr. Hayter

provided no such table. I did, and Mr. Hayter wisely approved of it amongst others he adopted from the same source—the original of every table in this section of the vital statistics of Victoria.

The particular table desiderated was devised by me for the very purpose of settling the most interesting of all the phthisis statistical problems to a young Colony—its etiology and possible prevention. To work out the forethought, I had to obtain a special grace from the Minister, and had also to personally supervise the clerk, who worked extra office hours in my own pay.* Not even a little clerical help could be got from the State, no more than credit from the learned Actuaries for the labour. Not that I cared for "*kudos*" coveted by others. Neither would I allude to

* The data were not even in the keeping of the Statist, but were in the charge of the Registrar-General; so that Mr. Hayter had not an opportunity to show his usual courtesy, greatly to his own regret. The gentleman from Mr Hayter's office, Mr. Fenton, who skilfully collated the details, had to go over 68,000 medical certificates of death for the items, compactly formulated in the following table.

the work now were it not to enlighten Actuaries, and direct them where they can find the information they seek.

Of the vast value of that knowledge one fact alone must here suffice, as it bears on the denied increase of the complaint in Victoria. In 1871, in the total deaths from phthisis of 841, only 81 were Victorian born; but in 1880, out of 1,175 deaths, 343 were so. That appalling fact has been before Victorian Actuaries and publicists for some time, and none dare try to refute it. The credit of bringing it to light is due to the unfavoured amateur. The only reason for adverting to the fact now is because of its great importance to this inquiry.

It will, moreover, be observed by the following table that in the period reviewed, 1871-1881, there were, in all, 11,354 deaths from phthisis in Victoria. Of these, 2,725 were amongst persons born in Australia, the greater part, or 2,234, being Victorians. The rapidly-increasing proportion which this number bore, and still bears, to the population living at the time will be presently considered.

Deaths from Phthisis in Victoria, 1871–1881, of Persons born in Victoria, Tasmania, and other parts of Australia.

Year.	Total Deaths from Phthisis.	Deaths from Phthisis of Persons born in				Percentage among Australians.
		Victoria.	Tasmania.	Other Australian Colonies.	Total Australia.	
1871	841	81	7	13	101	12·01
1872	876	97	19	10	126	14·38
1873	945	129	19	16	164	17·35
1874	1,011	137	21	21	179	17·70
1875	1,027	209	15	25	249	24·24*
1876	1,010	150	16	27	193	19·11
1877	1,088	197	30	27	254	23·16
1878	1,124	258	23	35	316	28·11
1879	1,058	296	22	28	345	32·60
1880	1,175	343	30	34	407	34·50
1881	1,199	337	21	33	391	32·61
Total	11,354	2,234	223	269	2,725	—

If the table afford Actuaries the required information, they are one and all again welcome to it. When I began inquiry I found in the new Colony "an interesting

* The effect of measles in increasing the death-rate from phthisis—as in 1875—is explained by the fact that when that fever attacks the phthisical it is fatal, the death being rightly set down to the graver complaint. Dr. E. Symes Thompson referred to this outbreak of measles as affording proof of a "pandemic disease

"field for scientific inquiry afforded by the "records of the Colony of Victoria," public and hospital, precisely as do the *Institute of Actuaries*, whose labours I trust mine will now facilitate.

Dr. E. Symes Thompson was surprised to find how small was the number of those who go out to Melbourne to die in the hospitals of consumption. Yet that distinguished physician has often been adduced amongst the high authorities who were opposed to the "exceedingly strong "deductions" drawn by me from the very same statistics that are now brought forward in the London Institute of Actuaries to startle, or amaze, and alter every pre-formed opinion. *Festina lente* may be a belated traveller.

---

wave," drawn on by sun spots somehow. It can be accounted for in a plainer way. The "*diademic*" began at Adelaide, where the first case was imported in the immigrant ship "*Hesperus*," and from thence ran all over Australia, ending at Fiji. Thus it is seen how easily all the facts about measles and about phthisis can be accounted for whenever they are written of according to the laws of Nature, and not of esoteric language.

On the exploded notion, that the phthisis mortality in Victoria is maintained at a high average by persons coming to the Colony in the incurable stages of the disease, attracted hither by the fame the climate had gained for curing the malady, and who after coming are not cured, but remain to die, a word need only now be added. The idea clings to some minds—nay to many minds, indeed to most minds—that so it is, with a tenacity showing how deeply rooted is the mental cancer of a false first impression, which, though cleanly cut out by the knife of the surgeon, still erodes and ulcerates.

For a dozen years past the vital statistics of Victoria plainly prove that only a small number of the recorded deaths from phthisis occur in new comers. By minute inquiry it is found that only some 3 or 4 *per cent.* so increase the death-rate. Leaving out the native-born whites, whose deaths from phthisis were $34\frac{1}{2}$ *per cent.* of the whole number of deaths from that disease in 1880; in the remainder, the period of residence far exceeded the stated duration of the disease, which

therefore *must* have been contracted in the Colony.

When attention was called to the extent of phthisis in Victoria, before 1870, it was commonly said to be due to invalids coming from England in a dying state. To learn if it were so or not, inquiry was made of the Registrar-General, then Mr. W. H. Archer, for data, but they were refused, the Minister, Mr. Attorney-General Michie, "dressed in a "little brief authority," not allowing access to them; and his "fantastic trick" in arbitrarily refusing a sight of the public archives was the mistake. Recourse was then had to hospital records, and these quickly dispelled the illusion. They proved that not a tenth of the deaths were of new-comers, and all that has since been done by the elaborate figures of later years only fortifies the conclusion.

Even if all the cases of which no particulars could be learned were added to the list certified as new-comers, the two taken together would not alter much the aggregate result. This test was first applied in 1870. (*On Phthisis*, pp. 28, 53.)

In the *Year-Book* for 1882 Mr. Hayter states how he, in 1873, ventured to predict that phthisis would be found to be on the increase in Victoria; and he now compiles elaborate tables to bear him out. But surely Mr. Hayter cannot forget how, in 1870, that increase was the argument of my work—its very *raison d'être*; or how I had to obtain an order from the Legislative Council on 1st November, 1870, to authorise him to furnish me with the statistics required to prove the fact. It was while occupied in taking out those returns for me that Mr. Hayter found the reasons for his prediction. I had to take the deaths from phthisis in the total deaths from all causes as my test; but Mr. Hayter, three years later, was enabled to take the deaths from phthisis in the population at given ages; and though his is the truer method, mine led to the same result, namely, that the disease prevailed largely, was increasing, unaffected by climate, and that about one in three of the adults who die in Victoria die of phthisis. But it is of far less consequence to know who first drew attention to the

increase of phthisis, than it is now all important to show how the disease may be lessened. This is the practical work on hand, that must be guided by theory.

That phthisis has greatly increased amongst native born whites in Victoria during the last decade is easily shown. The following is the proportion the deaths from phthisis at the ages 15 to 25 bore to the numbers living at the same ages, giving the deaths in every 10,000 of the Victorian born portion of the population at those ages, as ascertained at the two census years 1871 and 1881 :—

Deaths from Phthisis, 1871 and 1881—Ages 15 to 25.

Year.	Persons Living, 15 to 25.	Deaths from Phthisis, 15 to 25.	Deaths from Phthisis per 10,000 living, 15 to 25.
1871	103,466	81	7·9
1881	183,775	181	9·9

The 81 in the first line include all ages for that year. The exact number would be about 74 at the specified ages, and hence the contrast between the two years should be so much the greater.

Here, therefore is exact proof that phthisis has increased during the census decade from 7·9 to 9·9 per 10,000 living, of the Victorian born, at the young adult ages of 15 to 25 years.

In the next place, here is a tabular view of the numbers who died during the year 1880 of phthisis in Victoria. It will be seen how few deaths occurred from it between 10 and 15 years of age; and that between 15 and 25 every third death, and from 25 to 35 more than every third death, was from phthisis. The vast majority of the population at those ages, 15 to 35, were native-born.

Deaths from Phthisis, 1880—Age at Death.

Ages.	Number who Died of Phthisis.			Percentage of Deaths from Phthisis to those from all causes.
	Males.	Females.	Total.	
10 to 15 years	5	7	12	5
15 ,, 25 ,,	104	149	253	33
25 ,, 35 ,,	140	136	276	36
35 ,, 45 ,,	116	109	225	23

It was quite impossible to determine these proportions until the census returns of the ages of the population were completed; but

now that they are available, they confirm all I have affirmed during the last dozen years.

The table below indicates the increasing rate of phthisis in young Australian adults during the years 1879, 1880, and 1881.

Deaths from Phthisis in Victoria in 1879, 1880, 1881, *of Persons born in Australia.*

	Numbers.		
Ages at Death.	1879.	1880.	1881.
5 to 15 years ...	29	23	30
15 ,, 25 ,, ...	157	209	290

Hence, while few suffer from phthisis between 5 and 15 years of age, after the turn in youth a sudden rise occurs in the number of victims. This point will be again referred to when we come to consider how its happens that contagion is most apt to be caught by breathing germ-laden air after puberty.

But there is further proof of the increase of phthisis in Victoria during the decade obtainable from the deaths from it per 10,000 of the population at the different ages, including Victorians.

This proportion is clearly set forth in the following instructive table, to the details of which I would beg to call the reader's careful

attention. It shows how phthisis has increased at the younger adult ages. At 15 to 20 from 7·69 to 10·23; at 20 to 25 from 16·99 to 22·28; and at 25 to 35 from 21·32 to 26·45 are ratios giving a large increase at each lustrum.

Deaths from Phthisis per 10,000 living at different ages, in Victoria.

Ages.	Victoria.	
	1871.	1881.
Under 5 years... ...	1·71	1·58
5 to 10 ,, ...	·56	·94
10 ,, 15 ,, ...	·93	1·85
15 ,, 20 ,, ...	7·69	10·23
20 ,, 25 ,, ...	16·99	22·28
25 ,, 35 ,, ...	21·32	26·45
35 ,, 45 ,, ...	20·02	25·16
45 ,, 55 ,, ...	21·85	25·13
Above 55 ,, ...	22·23	21·76
At all ages	11·49	13·80

Therefore, during the census decade 1871-1881, phthisis *has* increased both in the total number of deaths from 841 to 1,199, and also in proportion to the population living at the different ages, and even at all ages from 11·49 to 13,80. This is marked enough.

The following are the rates for males and females in a very suggestive form :—

Deaths from Phthisis, per 10,000 living at different ages.

Ages.		1861.	1871.	1881.
Females.	15 to 20 ...	14·07	12·37	12·50
	20 ,, 25 ...	18·95	19·28	20·77
	25 ,, 35 ...	24·76	22·62	26·36
Males.	15 to 20 ...	7·72	5·71	6·88
	20 ,, 25 ...	12·23	18·75	20·69
	25 ,, 35 ...	16·53	22·21	27·94

It cannot be urged that the young women from 15 to 25 years of age have come into the Colony to die. They were chiefly native-born. It might be argued that females are more prone to phthisis than males. But will it not more accord with the newer and truer etiology of the disease, and therefore be more rational to infer, in the light of that better knowledge, that young girls are hired out to domestic service, and in their avocation of cleaning bedrooms are brought into contact with the active contagion of the disease ?

The remarkable proclivity to phthisis of domestic servants is doubtless wholly owing

to the frequency with which young girls fresh from open-air country life are, in their daily household work, brought into contact with infective particles thrown off from ulcerating lungs affected with the specific malady—particles all the more deadly because unheeded, or deemed innocuous, and therefore never dreaded. Not that ordinary domestics are occupied nursing or tending invalids; but cleaning bedrooms, and consequent inhaling dust of dried sputa, must obviously be a daily occurrence. In such young women phthisis is commonly attributed to cold, or badly-ventilated, dank sleeping rooms, but seldom or never to the really potent agent, direct contagion. No effort is ever made to intercept infective particles from phthisical sputa floating in the air, or to ensure their chemical destruction. Indeed, he would be talked about as a fussy enthusiast who would propose to do anything so pragmatically absurd, and might even be shunned in practice for his prodigious nonsense.

It will not often greatly avail to interrogate sufferers themselves, for they indeed can rarely

tell, and seldom do they even guess, where they caught contagion ; many, indeed, hardly entertain a lurking suspicion of the real source of their illness, so insidious is the virus, and long latent ere it excite attention in the earlier phases of the affection.

The Jewess is rarely tubercular. But Jewish maidens never serve the tainted Gentile, to breath tubercular breath, as we try rabbits in experiment.

That the mortality from the yearly increasing phthisis in all Victoria does not yet quite equal that from the same disease in all England is true. But can Victoria and England be fairly compared for this criterion? One may fairly trow not. Is there any one English county where run upon run of 10,000 to 50,000 or 60,000 acres are occupied by a handful of nomads? Why, the whole territory has not so many inhabitants as one fairly big old country city. The comparison is sheer nonsense and preposterous. A far fairer one is to compare all England with Melbourne and suburbs, an area twenty miles across, occupied by under 300,000 people.

That comparison is now all the fairer, since it is shown that *all* the cases do not occur in Melbourne. The overgrown young city, a veritable Cornstalk, has its own phthisis, as has already been abundantly shown. Therefore, the writer adheres to his original propositions—that, at given ages, phthisis is as prevalent in Melbourne and suburbs as in England; that the disease is increasing; that the native youth enjoy no immunity; and that, whenever the specific cause of the disease comes into operation, climate is impotent to control it. These were for the time truly "exceedingly strong deduc-"tions" from the meagre data at hand; but they were among the forecasts potential for good of the century.

In applying a final test to the original inference, the proof need be no way theoretical, but a practical criterion, based upon an admitted fairness in comparing Melbourne and suburbs with all England in their respective death-rates from phthisis. Some people object to the equity of the comparison; but all who have best thought out the matter admit the

validity. The number of persons to every square mile is equal in each country, and the occupations of the inhabitants are in both very similar. In the nearness of person to person the parasite causing phthisis can more readily propagate. That is how density of population fosters contagion.

Finding, then, in Melbourne and suburbs—"Greater Melbourne," with its seven cities full of factories, its villa-clad hills, open spaces, parks and gardens, its pastoral plains and farming fields—a miniature England, peopled by the same race, in nothing altered in habit or manner of life, save and alone the sun that brighter shines over them, their relative conditions in regard to the prevalence of phthisis will now be compared, not to sow disputation, but to reap wisdom.*

In the first place, the number of adults living at the ages prone to phthisis in Mel-

* The reader who would require fuller detail about the comparison above made will find it in the original essay *On Phthisis*, 1870, pp. 44 to 48; in the *Victorian Year-Book*, 1875, p. 136; and in the *Australian Medical Journal*, 1877, p. 359.

bourne and suburbs is far over one-third of the whole number in the Colony. Thus, of 183,000 between 20 and 25 years of age, 73,000 reside in the metropolitan district. Of the total population in the Colony of 374,000 between 15 and 45 years of age, 139,000 live there. In that central population is found the material for the phthisis parasite to prey upon. That material does not require to be drawn from England, nor yet from our inland towns. Esoteric reasons account for facts that are always obvious enough to everybody except the philosophers.

From those population returns of the last Victorian census, 1881, the phthisis death-rate in Melbourne and suburbs will now be compared with the corresponding death-rate in England, at the same ages, in 1871, the latest English census year available. And probably the English phthisical death-rate has not since then varied so greatly as to invalidate the comparison. Even the many who go abroad to end their days must be but a mere handful of the multitude whom poverty compels to stay at home in England to die.

Deaths in adults at 15 to 45 years being alone compared, the following list is limited to them :—

Deaths from Phthisis per 10,000 living at different ages in Melbourne and Suburbs in 1881, and in England in 1871.

Various Ages.	England.	Melbourne and Suburbs.
	1871.	1881.
15 to 20	23·33	18·90
20 ,, 25	33·33	34·81
25 ,, 35	41·33	39·48
35 ,, 45	39·98	41·00

The foregoing table needs no comment; the facts speak for themselves, and the figures cannot be altered by all the Actuaries in the world.

The details thoroughly confute the conclusions come to in 1877 by the Medical Society of Victoria,* and ordered to be "forwarded to the principal newspapers in the "Colonies and to the medical journals of the

* *Australian Medical Journal*, December 1877, pp. 365, 378.

"United Kingdom." Will the learned Society as widely give the correction? "In "medical research, nothing comes between it "and Truth." That is the only prestige to uphold, the only pride worth upholding.

The chief deduction which the Medical Society of Victoria may now rectify is their fourth, affirming that—

> The reduction of the mortality among young persons is to be explained by a comparative immunity among those born in the Colony.

The false confidence inspired by that dictum wrought for a time great mischief. It gave a semi-scientific backing to a popular delusion. To eradicate that impression, the invidious duty was thrown upon me of seeming "to run "down the Colony." The absurd idea got about that I wished to prove that this climate was bad for phthisis, whereas I affirmed that neither this nor any climate was good or bad for the disease, which did not depend on climatic influence in any way, but only on a specific germ poison that could operate in any climate. In this view I am now fully con-

firmed. The climatic idea of the genesis of tubercle and of phthisis is exploded.

It has been found by experiment by Dr. Koch that the bacillus of tubercle requires a temperature equal to that of the human body for its growth. Now animal heat is the same in all climes, hot or cold, dry or moist, rural or urban; it is only raised or lowered by the action of fungoid organisms multiplying in the blood or tissues of the living body. Therefore, whenever the tubercle parasite lodges within the body, the invader is cherished by the warmth and refected by the protoplasm into activity, regardless of outward states of climate. Hence the axiom was affirmed that "Whenever the specific cause of phthisis "comes into play, climate is impotent to "control it." That axiom was set down at the time as quite untenable. Yet it is now every day found true.

Even in the pure cold air of high regions, once thought aseptic and antiseptic, phthisis is as readily caught by the healthy servants waiting on the sick as it is caught on the moist warm plains below. After giving sundry

examples of contagion conveyed to attendants, native and English, upon the sick at the now frequented and often crowded fashionable resorts for phthisical invalids, Dr. Burney Yeo remarks :—

> If the infective character of tuberculosis were generally recognised, and the tubercular nature of pulmonary consumption generally admitted, mistakes of this kind would hardly be committed.

And yet this was the very grand mistake which has been committed in Victoria, where the pure dry *hot* air ozonic was thought to be equally curative and preventive. Verily, one might well repeat and apply the remarks lately made by Dr. W. R. Thomas, in a lecture on Phthisis and the theory of infection, delivered to the students of clinical work at the Sheffield Medical School, that—

> We are apt to discard all new theories and all new methods of treatment, and we are not alone. Those who have gone before us have done likewise. We see theories propounded by some years ago, and then not received, brought forward in our day with additional explanations, and at once accepted by the profession. Owing to our advanced knowledge, we can now see what could not be appreciated before; and, in the interim, thousands have died whose lives might have been saved.

Having shown the peculiar importance of the inquiry, in which young Australians ought to feel an especial interest, I shall now offer a few remarks on the nature of the phthisis contagion. In so doing, I shall repeat what I wrote in 1876 and 1879, illustrating it by some observations by later writers, by whom it is confirmed.

If, then, it be neither the atmosphere over the roof, nor the ground under the foundation, but altogether air within the domicile that contains the cause of phthisis, the potent agent is so far brought nearer ken in ultimate analysis. Let us, therefore, try to follow to its lair this hiding evil, and test whether it be ever found in catarrh, pneumonia, imperfect respiration, scrofula, impaired nutrition, or other malady or defect with which it is usually coupled; or whether it be not but a germ in its own kind.

Without adding a word more than need be on the relations which pneumonia, bronchitis, and tubercle bear to phthisis, a remark may here be offered on the nature of the infecting particle, now admitted to be the

special irritant, to set up morbid action within the air-chambers that end in that disease.

The remarks may seem very technical, but this cannot be avoided in a point of pure pathology.

That the infective organisms act on epithelium within the air-chamber rather than on that in the ultimate bronchiole, is explicable by the anatomical character of the alveolar epithelium being nearer allied to endothelium of serous membrane than to the columnar form on a mucous surface. They will there, moreover, meet with the adenoid glandular tissue peculiar to every pathological requirement in the disease.

These distinctions are clearly indicated in papers in *The Practitioner* on pulmonary pathology by Dr. Hamilton, whose views on the intra-alveolar origin of tubercle by accumulated epithelial scales were given in that journal shortly after I had offered views quite similar here. Dr. Hamilton explained the accumulation by a proliferation rather than as a mere relic, as I had done. In acute bronchitis, after the dead epithelium has

come away, basement scales in the finer or ultimate branches of the bronchi do not proliferate; and as these scales are continuous with the layer of pavement epithelium of the air vesicle, the same explanation must by analogy apply.

It is hence clear that the giant cells are only ordinary old worn-out epithelial cells cemented together, and not new growths.

This is the view taken by Dr. Shepherd, who in 1877 wrote:—"Interstitial, extra-"alveolar growth is not the commencement "of ordinary consumption; that these growths "play but a slight, and that a secondary, "part in phthisis; and I shall hold, with "authorities equal at least in reputation, if "not in number, to those who support the "contrary, that the pulmonary consumption "of this country consists primarily in intra-"alveolar changes." That is the precise view put forward by me in the 1876 opuscule. As I concluded, Dr. Shepherd agreed that giant cells were only ordinary epithelial cells in another form. That form seems to me to be no other than the *tubercle corpuscle* which

was described by Lebert as unique. It was "made up of small, irregular, angular cells "that were nothing else than dried, often "fragmentary cells which had lost their "vitality." Lebert was thus on the very verge of the discovery of the true nature of tubercle, requiring only the idea of the parasitic organism to make it complete.

In his lectures, 1878, Dr. J. Henry Green likewise described tubercles as formed by epithelium accumulated within the alveoli, and containing cells and granular or amorphous material, but he could not speak with certainty of their exact nature.

As late as 1882, Cornil and Ranvier, in their text-book of pathology, submit the cause of tubercle as still *sub judice*. In 1880, as already mentioned, Professor Cohnheim looked forward to the day when the tubercle corpuscle would reveal a minute organism. In 1881 Dr. Creighton, dubious, held the anatomical definition of tubercle useless, and trusts to the ætiological. Since then Dr. Koch has demonstrated what Dr. Creighton, of Cambridge, a year before doubted, and what

I had, six years before, plainly to them all described; for such is the zig-zag march of medical science.

In that incertitude, painful to all who had to deal every day with a disease of the true nature of which high science could tell nothing, nor offer one hint how to cope with it, fresh investigation was required. Hence my venture to explain how a nodule, or tubercle, comes to be formed within the air vesicle by the impacted *debris* of the wasted epithelium scales left there after they had been blighted in being robbed of their protoplasm by the suction of these parasites; and that the giant cells and other constituents of the tubercle, are relics, and not new products of a morbid cell growth.

That account best agrees with the usually progressive character of true phthisis. Of the self-limiting nature of the disease, occasionally observed, as lately shown by Dr. Austin Flint, it will also afford an explanation.

How these epithelial cells so wasted accumulate to form infinitely innumerable foreign bodies in the alveoli, irritating surrounding

tissue, I endeavoured to explain by the action of the phthisis micro-parasite, or infecting organism.

The morbid process must be observed within a single air-vesicle. For the whole process of lung impaction is but a repetition and aggregation of what goes on in one chamber. The physiological botanist will analyse the contents of a single grape to learn the nature of the parasite blighting a bunch.

These are admittedly abstruse and difficult questions even for a refined pathology to decide; yet they are all essentially necessary to be understood before a clear idea can be formed upon the subject.

Therefore this would be the place to describe the mode of action of the parasitic organism eroding the epithelium of the air-sacs into a *debris* that forms tubercle.

But this explanation having been given in a separate publication in 1876, I shall now beg the reader to refer to it in the appendix, where it is given in the same form. I shall, meanwhile, here offer some practical illustrations of the contagious action in phthisis.

The histological or cell changes met with in the lungs in pulmonary phthisis are essentially similar to, or rather they are identical with, those which occur in acute miliary tuberculosis, an admittedly infective disease.

May not our analogy go further, and accept phthisis to be equally infective? In phthisis, as in tuberculosis, the large nucleated elements in the alveoli are evidently the offspring of the epithelial cells which line the alveolar cavities. These contents of the air-chamber are accompanied by infiltration in the alveolar wall, a change in its adenoid tissue doubtless induced in the first instance by the irritant or infective particle acting on the protoplasm of the epithelium of the air vesicle, rather than in inter-alveolar or proper lung tissue, as was until lately held by many pathologists.

Here, then, there so far appears to be a connected series of morbid actions all directly connected with specific infection; and the question will naturally arise, can they be traced a step even further back? Dr. Green says:—"In describing the several lesions as

"inflammatory, I would again repeat that I "merely mean to imply that they owe their "origin to *some kind of injurious irritation* of "the pulmonary tissues." When infection becomes an auxiliary in the production of phthisical consolidation of the lungs, "the "infective particles are usually derived from "some pre-existing phthisical disease." All phthisis is inflammatory, but to apply the term "pneumonic," tends, Dr. Green adds, to mislead.

The pneumonia preceding or accompanying phthisis would therefore appear to have a special feature, allied to tuberculosis. Is not the differentiating element the septic virus? and the form the fungaceous organism endowed with special pathogenic property? And if tubercular virus, or phthisical material, be thus transferable from one locality to another within the same body, may it not likewise be transferable from one body to another through the atmosphere?

This transference of infecting particles is the mode of communicating phthisis most obvious with present means of judging. Virus

conveyed from a diseased to a healthy lung sets up therein the irritation and inflammation peculiar to phthisis.

This peculiarity may give a clue to the cause of the sudden increase of phthisis in native Australians approaching maturity. There may be unusual exposure to inhalation of specific virus. If phthisis were caused by cold and damp in ill-drained towns, it might be expected to affect all ages. But the disease does not appear to be induced by mere cold or damp in the absence of that special irritant already mentioned.

The crowding or huddling young folks together in sleeping-rooms and work-rooms facilitates the process of transferring virus very much as children catch ringworm. Nobody would hesitate to separate a ringwormy head from other heads; but who amongst us would dream of excluding a lung loaded with infecting irritant particles or bacteria in the blighted epithelium of diseased air vesicles? Nobody would be so infatuated, absurd, hard-hearted, and theoretically cruel. Yet an analogue of the organism is at

havoc on the mucous lining of the air vesicle as effectively and as communicable as is the irritant that sets going the peculiar irritation in the protoplasm of epithelium on the outer skin. The result produced cannot be equalled by any common inflammatory action. That much is certain. The means of preventing ordinary catarrh, or inflammation, do not suffice to check the action of the special irritant inducing phthisis.

Hence, all general conclusions drawn from the presence or absence in any country of common inflammatory lung affections from seasonal changes are utterly fallacious when applied in *a priori* reasoning about the ætiology of phthisis.

The older idea of the origin of phthisis agreed with the pathology that long held tubercle not specific, but secondary to pneumonia, tubercle pearl being in that view merely altered exudat left after an inflammatory process. It also chimed in with the still popular belief about catching cold being the ordinary beginning of phthisis, especially in enfeebled or broken-down constitutions.

That the initial irritant is the infecting bacterium acting on the epithelium of the air sacs, exactly as a bacterium is the irritant in ringworm* acting on the protoplasm of the nascent epithelial cells on the outer skin, is the more rational doctrine; and, moreover, if indeed there be merit in priority, a doctrine which was long and often advocated here ere it was known to be advocated elsewhere.

This was the principle that guided me in phthisis. The disease I viewed as a strictly local infection of the air membrane of the lung in the first instance. In this way we argue from analogy, as a sure method of reasoning, wherever we can trust to the unerring uniformity of Nature.

An infective agent in phthisis and tuberculosis had often been referred to by

---

* "On *Bacterium decalvans:* An Organism Associated "with the Destruction of the Hair in Alopecia areata." By George Thin, M.D. Communicated by Professor Huxley. *Proceedings Royal Society*, March 1881.

Dr. Thin says:—"The disease was at once and defi- "nitely arrested by a treatment designed to destroy the "vitality of any bacteria which might be present on the "surface of the skin."

pathologists; but none before me had given it a name. In saying what it was like, or belonged to, one was not over-bold. The blank in pathological knowledge and nomenclature was open to a venture. That blank I filled in and the result is of infinite practical value. It explains many points hitherto veiled in doubt. One of these I shall now offer in illustration.

Thus, no physiological reason had yet been given, though many efforts have been made to show why the upper lobe, or apex, of the lung is most liable to attack. And yet, with infection depending on inhaling an irritant particle, it is clear that the air vesicles most readily reached will be those most exposed to contagion. The air vesicles are carefully protected by the columnar epithelium of the minuter bronchioles hindering entrance to the smallest body. But if under relaxed states of the general system, the respiratory movement is carried on in an atmosphere laden with exuviæ from phthisical lungs, inhalation of their infective material and inoculation therewith will readily take place.

These considerations led to the idea of a true basis for a pathological classification of cases of phthisis into real and simulative, the latter arising from inhalation of merely mechanically irritating particles of dust, as in iron grinders, potters, colliers, cabinetmakers, and the like, in whom the disease readily subsides on removal of the mechanical cause; while the former, or true phthisis, comes alone from inhaled organisms that propagate themselves in the living tissues, and so cause the changes in the affected lungs to become progressive. Their secondary effects include cell infiltration between the alveolar walls, condensation of the connective lung tissue, vascular obliteration, and other structural changes known to the morbid anatomist.

This may have appeared mere speculative writing; but it was all-important for the question under consideration. That investigation was required was admitted by the best example. In 1878 Dr. Green, alluding to phthisis as *par excellence* an apex disease, frankly admitted, in reference to the reason why it is so, that "our knowledge on the

"subject is undoubtedly most incomplete;" while in his work on morbid anatomy of the same year, it was but conjectured that the infective agent may be a "minute organism" —that is, an organism as had been described by me in 1876. Such a body would necessarily exert a very different influence from a merely mechanical irritant; it would behave rather in the manner of a *contagium vivum*, or septic organism.

Hence it follows that, in explaining the necessity for free ventilation wherever human beings gather, it will not be enough to expatiate upon the need of renewal of air to replace that vitiated, or rather deprived of its vital part by previous respiration. It must be still more important to point out that in breathing pre-breathed air there is always the probability of inhaling the specific organisms of true pulmonary phthisis. Being so, then all idea about climate conferring an immunity from phthisis becomes the most pernicious doctrine that could be inculcated.

That specious delusion is all the more dangerous, unless it could be shown that any

condition of ordinary air is capable of oxidising or destroying those particles while they are active within the pulmonary alveoli, or in the outer air, after they have been exhaled, or otherwise extruded from the lungs. Of the existence of such a quality in common air there exists no proof whatever.

Oxidation does not kill living organisms; it only neutralises the virulent element that is a morbid accident of *some* bacteria. Oxygen attenuates the virus of bacteria in the open air, but not within the tissues of the living body. For this action a more potent agent is wanted. To arrest the eroding action of bacteria in the air-cells of the lungs an antiseptic is required that will not affect the oxygen of the respired air. This was our great desideratum that has only just been touched. To supply it will excel all that has ever yet been done.

The organisms of tubercle are as active in the cold dry air of Swiss mountains as they are in the dry hot air of the Australian bush; and they are no less active in either than they are in the crowded haunts of a large city.

The vitality of these infective particles in phthisis, external to their bodily nidus, may be equal to that belonging to typhoid contagium. That against either contagium peroxide of hydrogen, or gum-tree aroma, are inert, we have in this colony abundant proof.

The researches of later observers confirm the view taken of typhoid virus in a *Report of an Inquiry*, by the author, into the etiology of that fever; and it in turn threw a practical light on the specific contagium in phthisis.

Indeed, it was while engaged in working out the problem of the etiology of typhoid fever in 1874, for the Victorian Government, and laying down the principles on which the health authorities now act in checking the spread of contagion, that I was led on by analogy to trace the true etiology of phthisis. Not that I found, as Dr. William Budd argued, that phthisis was primarily a continued fever allied to typhoid, with lung disease as the sequel or dregs. On the contrary, I found phthisis to be from the beginning a

local infection of the epithelium of the air sacs of the lungs, gradually spreading from its nidus until it sets up constitutional irritation and its train of sequents, with a symptomatic fever akin to the septic fever of surgery.

Therefore the new theory of a local infection setting up irritation with general fever was more allied to *Listerism*. Indeed it was the first time that that principal was applied to clinical medicine.

Let us draw an analogy to illustrate this now all-important point in practical medicine, and in public hygiene.

In 1874, in the *Report* on typhoid fever referred to the writer remarked that:—

> To assert that typhoid fever is only contagious in a limited degree, is ambiguous, unscientific in theory, and perilous in practice. The popular and the medical notions are of the same vague sort. The difference does not lie in the mediate or immediate mode of communication. A deeper pathological distinction in theory and practice may be drawn. In contagion the virus is reproduced within the diseased body—true contagia having no other known source, unknown as entities except in connection with diseased bodies. The meaning differs from the primary or etymological sense of the term; but it is the best medical definition consistent

with known facts, and, if invariably adhered to, would remove a great source of confusion from both lay and professional minds, and tend to clearer views on the causation of acute specific fevers.

That was the writer's proposition in 1874. Unhappily his professional critics on the Central Board of Health did not approve of the innovation. In their *official* gloss to the text they rejected the idea of "'contagious,' "in the sense Mr. Thomson puts on the "word." And yet that sense is the very sense which Dr. Burney Yeo* now puts upon the word, and admonishes the members of the medical profession to follow his example, because of the great practical utility of the new definition. Modern teaching has widened and corrected the conception of "contagion;" and in the new pathological doctrine it becomes "quite possible that, under certain "given conditions, consumption may be a "contagious disease." Hence we in this

* Clinical Lecture on the Contagiousness of Pulmonary Consumption. Delivered in King's College Hospital. By I. Burney Yeo, M.D., F.R.C.P., Physician to the Hospital. *British Medical Journal*, 17th June, 1882.

Colony were several years in advance of the old world ideas on those matters.

This wider definition of "contagion" is also borne out in the difference between true and false tubercle. Dr. Hippolyte Martin, of Paris, finds that inoculation with true tuberculous matter is alone capable of reproducing true tuberculosis, lesions from non-tuberculous matter being only pseudo-tubercle. The anatomical structure of both true and false tubercle appears identical under the microscope, but in a series of inoculations they differ. True infective tubercle is reproduced in an indefinite series, but the pseudo-tubercle is perfectly innocuous; there are no bacteria to propagate. Dr. Burney Yeo further observes:—

> Consumption is by no means contagious in the sense which is ordinarily and popularly attached to that word. But the modern methods of experimental research are, doubtless, destined to widen our conception of "contagion," as they have widened and corrected our conceptions in respect of many other pathological doctrines.

The primary question therefore resolves itself into this:—Are we to cling and adhere

to the old limited vulgar idea of contagion popular in the pre-scientific era, or are we wisely to expand and open up at once both the idea and the connotation of the term, so as to embrace by it both the facts of induction and of demonstration? That is to say—Are we to define "contagion" according to the facts of nature, or agreeably to the antiquated teaching of the schools? Therefore, the perfect analogy between the argument for contagion in typhoid fever and in phthisis is a practical one, and not merely theoretical.

The influence of climate acting directly upon the human body to produce typhoid fever was asserted to have been practically shown in Australia last summer, but the fallacy was quickly exposed. Fever prevailed in many places; the season was everywhere hot and dry; but the heat and drought did not create fever without the mediate action of specific bacteria.

The infecting organisms that long retain vitality, and that have for years bygone been thrown broadcast about in a belief of their innocuous nature, were only more freely than

in wet seasons blown about in the dry hot air, under a glaring sun from the arid parched ground, without a blade of grass, or any kind of active vegetation. That was all; as is indeed in my oft mis-read *Report* fully enough made plain to any mind.

That phthisis is never, any more than is typhoid fever, of mere climatic origin is the obvious corollary. The specific bacterium is unknown except as it is derived from a diseased body, and cannot be conceived to originate under any circumstances *de novo*, as that organism would have to originate if climate created the disease which depends upon its exulcerating action.

I am often asked where the primordial germ came from, and whether like conditions for its *de novo* generation would not now generate it afresh. But of those conditions we know not anything, any more than we do of the conditions under which a turnip seed appeared upon the earth. All we do know is that without turnip seed we cannot grow turnips; that without tubercle germs we cannot breed tubercle; and that both tubercle

and turnip seed have been imported. That for us is primeval enough.

If, then, the real active cause of true progressive phthisis be a septic germ, from whose invading growth Victorian natives, black or white, have no immunity, it follows that if contact with the destroyer were stopped the fatal effect would cease.

The little organism differs in size but not in fatal effect from the banished *acarus* that long preyed on ovine life in this Colony, causing its own peculiar wasting. Yet if one parasite were caught in the pelt of a sheep, the fact would ring consternation over Victoria, and stringent penal laws would be passed for its destruction, though myriads of the other, thriving in the morbid lungs of the sheepowner's children, excite no notice. Zymotic germs of every other sort are forbid to enter the Colony, but septic particles of phthisis are freely invited.

Hence the cause of the rapid disappearance of the blacks before the whites, whose approach marks the onset of phthisis amongst the Aborigines. The latter had no phthisis

until the white man came amongst them inoculating. Their tubercular phthisis is in every way identical with his. If the white man's phthisis be hereditary, as some contend, the black man's was not. But the phthisis in both races, like their syphilis, is alike; pathology cannot define any difference. Hence the one race reads a lesson to the other. It would be absurd to affirm that syphilis was of climatic origin among the blacks; it is unscientific to maintain that phthisis so begins.

That the most fatal malady affecting the Aborigines belongs to the same order as the most deadly disease amongst white natives in Victoria is now plain.

Inquiry on the spot into the cause and nature of the destructive lung disease at Coranderrk revealed beyond doubt tubercular phthisis, spread by contagion. The spat-on earthen floors were sodden with phthisis sputa. The people breathed dirty air from neglected bacteria. The huts looked clean, but the air was germ-dirty. In that one factor lay the whole history of the propagation of phthisis among the Aborigines.

The protectors of those poor people will, no doubt, now take care to prevent this mode of infection in future. They will also take good care to say to whom they rightly owe their instruction. For in that "sort of things "it is the manner of men first to wonder that "any such thing should be possible, and "after it is found out, to wonder again how "the world should miss it so long." The Board of Protectors are grateful for scientific insight given to them as was of old the Central Board of Health, or as is the guiding daily paper they emulate. To act on admonition is the nobler recognition. The explanation given was not the climatic one that was required or hoped for. It went straight to the truth. It lent to neither side in a wild conflict of opinion, and from both sides it obtained tacit respect. The etiology of phthisis in black or white has no politics. They all alike believe in the "*crotchet*" now since it has been found to be in agreement with the highest science of a renowned authority. This assures them that they were not misled by an enthusiast, who

had the ill-fortune, fatal to popularity, to think awhile in advance of the hour.

In many patients, where the inception of disease was evident from inspection of them on the station, the fatal end at the Melbourne Hospital only verified the earlier diagnosis, by necropsy by the hospital pathologist, of lungs packed with well-defined tubercle, or emptied into cavities, accompanied by all the usual morbid changes in fatal phthisis.

Therefore the inquiry into the cause of phthisis amongst the Aborigines on Government stations cleared away one mystery long hanging over Coranderrk.*

---

* In the *Report* of the Board for the Protection of Aborigines for July 1879, it is stated that "lung disease "is the chief cause of death among the Aborigines, "who, when once affected, very seldom recover. The "Board has recently called the attention of the Govern- "ment to the serious mortality among the natives, "and it also contemplates calling in the aid of Mr. W. "Thomson, of South Yarra, who has devoted much "attention to this branch of disease."

The Board's dubiety about the nature of the fatal lung disease arose through a statement in an official letter from the Chief Medical Officer to the Hon. Chief Secretary, in which the writer, alluding to the "awful "mortality" at Coranderrk following an epidemic of

The Coranderrk station stands high on a sub-Alpine plateau in the heart of the gum-tree forest, where, according to modern teaching in a vague way, the balsamic air ought to be highly aseptic, and a sure preventive of the action of the septic bacteria. Yet when the specific parasites are carried thither they are quite as active as they are in the crowded towns, where the air is laden with animal exhalations. For example, when a gentlewoman in advanced phthisis went up to reside near the station for the benefit of that aseptic air, she did not recover, but remained to die, and in gratitude left some gowns and dresses as a legacy to a fine healthy young aboriginal girl, to whom she had taken a great liking. The poor girl wore the garments of which she was proud, and ere many months were over fell a victim to

---

measles, added that, of thirty-one deaths, "fourteen from "pleuro-pneumonia and chest disease point but too "surely to the damp floors of the huts as their cause. "Persons attacked by such diseases have scarcely a "chance of recovery in such hovels." But measles had also been fatal at other stations, vaguely-defined chest disease being again the complication.

phthisis. The ozonic air of the tree-clad mountain neither cured the disease in the sick nor hindered contagion from passing to the healthy. Perhaps the difficulty may be met by pleading that at Coranderrk the climate is cold and moist, not dry and cold. But what of the redolent balsamic air?

It is often held that phthisis cannot be contagious because it is not shown to be so in the Brompton Hospital. But there no dried sputa float about in the air breathed by nurses. There is more virus dust in one small bedroom in private life, or in a native's hut, with a phthisical invalid, in a single week, than in all the wards and waiting-rooms in the Hospital in a whole year.

A like fallacy occurred at the London Fever Hospital to prove typhoid not contagious—or, rather, as it ought to be called, transferable, or communicable, or catching, from sick to healthy. But there, again, no soiled linen is given a chance to spread disease, as we daily see in private life. Typhoid is a dirt disease; but the dirt that spreads the fever is no ordinary, but a specific, dirt. So is it with

phthisis sputa, that dirty the air with specific phthisis poison, even amidst much surrounding ordinary domestic cleanliness.

Nicotine fumes in tobacco factories have a curative power over phthisis, and by an action that must be of a purely local character. The dust or vapour from the leaf being uniformly diffused throughout the works, when a phthisical person enters the medicated air the nervous system usually soon becomes inured to the narcotic, but not so the lung parasitic organisms, which quickly succumb and leave the body they infest to regain normal life. Such appears to be the *rationale* of cure. That recovery does occur, is beyond doubt. The fact is attested in factories in this and other countries; but the action has never heretofore, as far as the writer is aware, been ascribed to an antiseptic virtue in the fumes of the drug.

The difference between an ordinary factory or workroom, with an atmosphere of common air, and such a place as a tobacco factory, with its air highly medicated by nicotine in fumes or impalpable dust, appears to be

simply this—that if workers in the former be exposed to the specific contagion of parasites, they may be infected; whereas, in the peculiarly medicated air of the latter, contagion from no sort of animal or vegetable parasitic organisms, external or internal, will ever take effect. Laden with nicotine, this air is aseptic and antiseptic of the tubercle parasite.

This may, perhaps, be further illustrated by the action of sea air on phthisical lungs in sea voyaging. That this air checks phthisis, is very generally allowed; but whether it act through abundant free oxygen, or by particular medicinal ingredients, contained in that air, is not clearly known. To bromine and iodine continuously inhaled in natural combination along with saline particles, in however minute quantity, the effect may be partly due, since these elements are peculiarly inimical to land animal parasitic life in every variety and form. Sea air would thus rank, *re* phthisis, as a specially medicated atmosphere, call it climatic or what you will.

The air filled with sulpho-arsenical fumes rising from the half-extinct crater of the

Solfatara, found effective in treating phthisis, as first mentioned by Galen, is supposed to exert a special local as well as a general tonic and alterative action. When more minutely examined, the local effect may be ascertained to be destructive of the parasitic organisms acting on air-vesicle epithelium in the manner already described. The same may hold true of the air near New Zealand geysers, said to contain chemical compounds from volcanic vapour.

An aboriginal, when ill of phthisis, left the Government station and returned to his old hunting-ground in the open bush, where he built for himself a bark hut to live in and breathe by day and night the smoke from burning gum-leaves and twigs until he quite recovered. From the humane squatter the invalid got all needful food; but the narrator affirms that without any doubt the cure of the disease was brought about entirely by the antiseptic reek. Had this poor deserter from his patrons been rigorously dealt with and sent back to his home at Coranderrk, he would have lingered a while in, for him,

a fine house with a highly-civilized chimney and chinkless walls, until it grew time to forward him on to die in the ward of a town hospital, wherein the antiseptic air-breathing had been tried and found to have no therapeutic value. Natural instinct is a wise physician, and well might have more practice; at least the untutored savage here taught the man of high art a capital lesson by example in the cunning of the healing art, but which enlightened prejudice was loth to follow. If willing to improve, we might here try the effect of hospital wards fitted for consumptive patients to live in, continually breathing specially medicated air; it would not be a costly experiment, although involving the humiliation of taking a hint from an aboriginal who brought his primitive clinic abreast the latest advance in bacterial pathology.

But, alas, there is nothing new in anything that is under the sun. In the *New Atlantis* Bacon says:

"We have also certain chambers, which "we call chambers of health, where we "qualify the air as we think good and

"proper for the cure of divers diseases, and "preservation of health."

On this passage the editor adds that, "This experiment has been tried, especially "by Dr. Beddoes, of Clifton, but without "any marked result. Some relief has been "obtained in cases of phthisis by inhaling "oxygenated air." Perhaps the trial might be repeated with better success under the newer knowledge of phthisis etiology. The good effect of such medicated chambers is shown by the antiseptic power over tubercle of the dust in tobacco factories; and a like influence was long ago related by Dr. Copland on workers in creosote factories. So also in chlorine and iodine works, and in gas-works. Those were all empirical observations, yet they none the less illustrate the germ theory of phthisis.

In relating the results of breathing germ-killing air in phthisis to incredulous pathologists, they try to explain away the apparent effect by assuming an error in diagnosis. Starting from the old familiar notion that phthisis is always a blood-gland disease,

originating in faulty nutrition in a system tainted with hereditary cachexy, and curable, if at all, only by change of climate, cod oil, and tonic medicines, these objectors to innovation argue that in seeming cure by breathing vapour instead of drinking drugs, there could not have been present any real lung disease. The plea is something gratuitous. Many of the cases had been already diagnosed as true phthisis by the ablest men in the profession. Not in every case has trial succeeded, nor will it in every case succeed. What plan will? But it has been often enough so far successful as to justify a steady effort to improve the method founded on the new etiology of the disease, and embracing the three elements of practical medicine, the preventive, the curative, and the euthanistic. The new etiology at last offers a hope that phthisis may not for ever continue to be a dire disease unpreventible in coming, nor yet an incurable one when it has actually come.

These examples enable us now to explain that *crux*, the baffling cause of the fatal

episode in Lænnec's Nunnery, where all the inmates were thrice renewed in ten years, owing to every fresh novice being at once seized by the deadly micro-parasite. Had the unseen destroyer been a visible viper it would have been slain; but ignorance of everything not plain to ordinary sight, however clear it might be to the mind's eye, would neither let the mite become known to science nor suffer the infector to be empirically killed. Are we a whit wiser now?

From those few instances, which, by the way, could be many times multiplied, it may be averred that phthisis may be more contagious in Victoria than it is found to be in England, and more like the form it assumes in the South of France, where a belief has always prevailed that the disease is contagious. But this idea has now vanished. The disease is allowed to be as contagious in England as in any country.

This realisation of my views widens out in every direction. Thus, in a recently published address on parasitic diseases, Professor Rushton Parker, of Liverpool, observes that:—

Tubercle is an infective disease, now known to be due to an organism. In the past one school held that it was a purely inflammatory process, while another always regarded it as a specific disease due to an infective virus. No doubt the histological phenomena of tubercle are consistently explained as inflammatory. But what causes the inflammatory changes? The very specific virus once thought to be antagonistic to the inflammatory view is now inseparably woven into it in the form of the tubercular bacillus, so admirably discovered by Dr. Robert Koch.—*British Medical Journal*, 17th July, 1882.

The impartial reader will here perceive from the above remarks that Professor Parker advocates the very same explanation of the nature and origin of tubercle which has been patiently advocated, in varied publications, by me ever since the beginning of 1876. For I have throughout maintained that the newer view will fully explain why, in its early stage of acute phthisis, the antiphlogistic and antiseptic methods of treatment must be combined. The reason for this twofold treatment could never be understood, unless under the revelation of the germ theory of the etiology of the disease.

Besides the histological proof that true phthisis must be transferable from sick to

healthy, clinical examples can also be given of the contagious action. These examples can only be derived from private practice, for in hospitals the details are impossible; or, at all events, they cannot therein be verified, but must be wholly taken upon trust or hear-say. Moreover, in a sparsely-peopled country like Victoria, the individual histories can always be more easily traced than they can be in older communities.

The following are the particulars noted in a few cases of phthisis in which contagion was evidently the origin of the disease:—

A young gentleman came to Victoria from England in good health. After living here for some time, enjoying active life, quite an athlete, he had news that a younger brother had got ill at school, and that the physicians urged a voyage to and stay in Victoria to arrest phthisis. On arrival the invalid went to live with his elder brother, the two sharing one bedroom. Within a few months the decease of the new-comer took place. In about another month the elder brother began to decline in health and vigour, and before

long spat blood. On seeking advice, and while stripping for examination, he said:—"If you find anything wrong with my lungs, "I have caught it from my brother." On being asked to explain, he briefly related the narrative as here given. There were very marked signs of phthisis in the early stage in the apex of the right lung. He had to give up his office work for a time and undergo very active treatment. The progress of the disease was checked; and though this happened several years ago, no actual disease has returned; yet the lad has never had the same elasticity and force of health that he had before his brother joined him in advanced phthisis, although he says that he has quite recovered.

A famous Victorian horseman, whose ringing voice calling the ostler will be by many well remembered, one day in the sale-yard spat blood. On asking him about it, he said he had done so several times lately, and added that he did not feel up to his usual energy. Shortly after he had another slight hæmoptysis, and sought advice, requesting at the same

time to have examined the lungs of the lady to whom he had been newly married. The patient was found in advanced phthisis, from which within a few months both husband and wife were dead.

The Principal of the Scotch College, Dr. Morrison, states how his brother left Melbourne in full health for a flying holiday trip to the old country. After travelling about, as an active healthy young fellow would travel for enjoyment, he took a passage in a grand ocean-going steamer on the outward run from London to Melbourne, and while on board had to occupy a narrow cabin along with a phthisical invalid in active disease. The poor sick lad so coughed and spat about that Mr. Morrison felt inclined to change cabins, but being good-natured and kind-hearted, he stood by his voyaging companion, feeling he had a right to do so much for charity. Not long after returning home he began to fall off, and ere long had to endure all the agonies save the closing pang of pulmonary phthisis. Dr. Morrison writes to say that neither he nor his brother nor

any of the physicians who were consulted had a doubt about the contagious origin of his brother's nearly fatal attack.

A farmer's wife living near Dandenong came under treatment for lung disease, having all the physical signs and general symptoms of active phthisis. The patient was about thirty years of age, and had been from early infancy in the Colony. Whether there was a family history of phthisis or not, or exposure to contagion, thereabout in plenty, did not appear.

Following her case came a near neighbour's child, who often visited and stayed awhile with the sick friend. This girl was about seventeen years old. There was here great emaciation and hectic, with dry crepitation over the whole right lung, back and front, and apex of the left, with inability to expand the thoracic walls, which, on drawing a deep breath, lifted or rather rose bodily, so completely was elasticity lost, and so abrupt the resilience. There also was incessant short dry hacking cough. For many months this girl's chest was kept under continual counter

irritation. During the while the patient inhaled antiseptic sedative vapour, and freely took bicarbonate of potass with her milk diet to check inflammatory tendency.

An excessive fibrin must be the local condition ever present under miliary tubercular deposit. That state of the blood could alone give rise to the pneumonic changes that constitute the secondary destructive process in lung tissue in the rapid forms of progressive phthisis so frequently met with in young people native to this Colony. These pneumonic changes in phthisis have made many pathologists doubt whether the primary lesion be not inflammatory. But if the newer be also the truer view, then the purely inflammatory hypothesis must have been a fatal error.

The newer view will, as I have already remarked, fully explain why, in its early stage of acute phthisis, the antiphlogistic and antiseptic methods of treatment must be combined. The reason for the twofold treatment could never be understood unless under the revelation of the germ theory of the etiology.

The germ is the irritant that sets up the inflammation, and it must be destroyed ere that effect can cease. With the germ killed the exciting cause of the disease is removed, and then the restoring power of nature brings recovery.

That mine is also the truer, as it is the newer, etiology is so far borne out by the results of clinical test-work in this and similar cases, for the girl is hale and well, with rosy cheeks and restored functions, active and lithe like other girls of her age who had stoical friends to go on with protracted treatment. Only under vacillation has like treatment in other cases failed.

Here, then, was a child who had been born and brought up in the very heart of the gum-tree forest, on a well-elevated site, in whom eucalyptic air daily breathed did not hinder the action of the specific contagion in pus spat up from lungs in well-defined phthisis, with which she had often come into close domestic contact.

In neither instance named had saturated subsoil, damp from want of underground

drainage, mountain site or swampy level, dry air or moist, bad ventilation, cellar home or factory toil, aught to do with creating disease, which owed its origin alone to conveyed contagion. What applied to those cases equally applies to all under an uniformly specific malady which cannot claim more than one uniform cause, to be outside the order of natural history.

What befel Mr. Morrison, and Mr. Curtis, Mr. R——, and the Coranderrk girl getting her visitor's disease along with the legacy of her garments, has befallen others, white or black, from phthisis contagion. A reason once is a reason ever, about septic phthisis germs in the sputa or in the air-cells, or specific pus of a common humanity. Similar instances of propagated phthisis where there was no inherited taint of the disease I have frequently met with, and many could be enumerated that in their results in recovery without leaving home would make the fortune of any noted health resort in the world. But multiplying facts builds no induction; we need not a heap, but an edifice. Therefore, only

a few more illustrative cases will now be given, though with the like it would not be difficult to fill a volume.

A lady in active phthisis came from the country to town for advice, and was welcomed to the home of a friend, whose daughter shared her bed or bedroom with the family guest. After a stay of some weeks the patient returned home in improved health. But not long afterwards the girl who had slept near the sick lady began to fail in health, have anorexia, with palor, debility, interrupted catamenia, loss of hair, dyspnœa, and short dry cough. After a while, the parents, becoming alarmed, asked for an inquiry as to the cause of this marked alteration in a young girl who was formerly active, robust, and healthy. On examination, besides the general symptoms named, very pronounced signs of acute phthisis in the first stage were found, dry crepitation with sibilant râles and long expiratory sounds being distinct. The chest was now wrapt in a counter irritant, and a pustular eruption kept out on the surface for nearly three

months; while during that time an alkalized diet was freely given, along with antiseptic inhalations, but no cough mixtures or tonics of any sort were allowed. The girl lost much weight during treatment. That the parents wisely did not heed. They knew the wasting had begun before treatment had been commenced, and justly ascribed the loss to the diseased action in the system, and not to any imaginary depressing effect of the medicines. These were given to control the local lung disease that, if left unattacked, would very quickly have completely destroyed lung tissue. That local diseased action on the lung, from direct contagion, being once arrested, the system so relieved of a local irritant soon rallied, and before a year the girl recovered, and is now, after four or five years, in fine health.

The same treatment in quite a similar case had arrested the acute action, when the patient was sent a sea voyage. To the sea air the arrest is ascribed wrongly. Had the arrest of the acute action not been secured before sailing, by very active antiphlogistic

and antiseptic treatment combined, the sea air would have aggravated the inflammatory action, as it did in the young gentlewoman from Ballarat, whose case is next to be related.

A very healthy family of many grown-up sons and daughters, living near Ballarat, in a healthy bracing air, invited an invalid relative to come and live with them, for change of air. The patient, a young lady, was suffering from phthisis. After staying with her friends for a few months she died of that disease.

The daughter of the family, who had devotedly tended and nursed her sick relative, sleeping in the same room with her, soon fell ill with marked symptoms of acute phthisis. The best medical skill in the colony was had, and a sea voyage advised. But within two or three weeks after sailing the patient died. The relatives confidently state their conviction about the contagious origin of the illness. The deceased was the only member of the family affected; and she had been, up till the time of receiving her sick visitor, strong and healthy. In her case the tonic and sustaining

treatment had been adopted from the beginning; the antiseptic or antiphlogistic undreamt of; with a rapid destruction of the affected organ. With a truer conception of the etiology, the malady in her case would have been arrested at the outset. But her physicians acted according to prevailing rule.

These two cases are instructive. In the former an eminent physician, irregularly consulted, predicted—prognosed—a rapid decline under any circumstances. But he had not been told the history of the case. That he inferred, and hence he "made a great "mistake." He was the happy physician who came in the decline of the disease. The patient was rapidly recovering before he was hurriedly consulted in a family panic. The patient had been freely exposed to active contagion by a cousin who died of phthisis about the time she fell ill.

A very well-known member of the Legislative Council of Victoria, whose interest in this question of the contagiousness of tubercle had been raised by his having observed facts bearing out the view amongst the cattle on

his station, drew the writer's attention to the following case:—

A man servant who had for many years been a strong, active, hard-working hand, married a delicate wife, who not long afterwards died of phthisis. The man soon fell off in health, and is now under a well-marked condition of the second stage of acute phthisis. A finer developed frame never was witnessed, nor a clearer example of direct contagion.

In all these cases it will be observed that the antiphlogistic and antiseptic treatment had to be combined. The disease was in the early stage when the septic germ had as yet only set up the irritant action, and before the resulting tubercle had advanced to the second stage of softening, with breach of surface, ulceration, or cavity. When this stage has been reached, which is the one when patients begin to go about seeking relief, a totally different line of treatment has to be adopted. It is now that the antiseptic inhalations alone are to be chiefly relied on. And it is at this stage when the boroglyceride becomes so eminently beneficial. Of this new antiseptic,

in its application to the treatment of phthisis, I shall relate only two or three particulars, that will suffice to illustrate the effects of its use, and leave the reader to judge of its probable value.

The first case in which I employed the boroglyceride was that of a gentleman from Tasmania, who came here for advice. His physicians advised him to reside for a time at Deniliquin; but there he grew worse. Another advice was to stay at Sandhurst, where the disease still progressed. On visiting me after that, I found, as others had done, very extensive disease of the right lung, with great debility, emaciation, hectic, cough, and copious typical expectoration. So ill was he that I felt inclined to say the case was hopeless. But so urgent was the patient to try any means likely to yield relief that I, at his earnest appeal, told him how to apply the boroglyceride spray and inhale it. After six weeks' use of the new remedy he returned for examination, when a marked improvement in both the general symptoms and physical signs had already taken place. The spray

has been continued, and now, after some five months' use, the disease is practically arrested, if not absolutely cured. Time however is yet required to find how far the improvement will be permanent.

A young gentlewoman came from the old country in good health to nurse a brother dying of phthisis. How he got ill is not known, as there is no hereditary taint in the family. Before his death his sister nurse was attacked. For eighteen months she was sent from place to place throughout the Colonies for change of air, but always growing worse. Three months ago I saw the case, and advised the use of the boroglyceride. Already there is a marked improvement. The patient is rapidly gaining strength; she can expand the chest and draw a deep inspiration without distress. The expectoration is lessening, and the physical signs are markedly better. The patient describes the feeling that when she uses the spray she feels as if something went direct to the tender part of the lung and gratefully washed or bathed it. That is the very sensation described by

those who have applied the boroglyceride wash to skin affections, as if the healing action were in both the same.

Since the above was written and in type another month has elapsed, leaving me an opportunity to again examine this patient. The closest attention fails to detect the least trace of disease remaining in the affected lung. So thoroughly has the deposit been removed that, had I not myself several times examined the case, I should feel disposed to doubt if an error of diagnosis had not been made.

A farmer living at Caulfield had become unable from phthisis to leave his house. His case was called hopeless. When I saw him the signs of extensive disease of the right lung were marked. I should have declined to treat the case had it not been tentatively with such a remedy as the boroglyceride, After using it for a few months the man is able to go about his farm, is gaining strength steadily, and promises complete recovery.

A young lady at Emerald Hill, who earned a living by teaching music, became so ill of

phthisis that she had to relinquish her means of livelihood. The left lung was much affected. A great variety of treatment had been tried without benefit. About two months ago she began to inhale the boroglyceride spray, and she is now able to expand the chest freely; is rapidly gaining strength, and the voice that was much affected is regaining its healthy tone. Whether she can rightly be called cured or not, or even in a fair way to be cured, she at any rate has greatly improved, and the treatment is being continued.

Of similar cases I can relate about twenty, in not one of which has the boroglyceride shown the least indication of having any hurtful effect on the general health. On the contrary, it seems to be wholesome, even nutritive. The old farmer, old, that is, at forty-five, cannot do without it. It appears to be equally grateful to all the other patients. It might be premature to pronounce definitely on the merits of the remedy from those few trials; but they afford to me proofs of utility. In physics one instance is

as good as a thousand; and why not equally so in scientific physic? It is not a nostrum tried in blind empiricism; it is a well-known and tested antiseptic used to destroy the septic agents in disease. I have had ample ocular demonstration of its efficacy in cutaneous disease, and I have had auricular demonstration of its efficacy in disease of the pulmonary surface of similar nature; and to me the auricular is equally as good a guide as is the ocular; so I shall proceed with the remedy.

Those few instances will serve to illustrate clinically the contagiousness of phthisis in Victoria, the bacterial nature of the disease, and the scientific method of curing and preventing it.

I shall now take up a very difficult and important point to settle, namely, the cause of the sudden rise in phthisis fatality in young Victorians just after they reach the age of puberty. To solve this question I must ask the reader to give a little attention to the numbers in the population living at the different ages, as ascertained by the census

in 1881. I promise not to make the calculations involved or irksome to those who do not care for statistics.

There are 862,346 people in Victoria, in 858,547 of whom the ages have been ascertained. Of these 499,199 are native-born. In the whole number, 330,246 are under 15 years of age, 100,212 between 15 and 20 years, 83,563 from 20 to 25 years, and 54,243 from 25 to 30. Hence far over a third of the whole population are under the ages most prone to phthisis, and only 183,563, or about one-sixth, at the most liable periods.

Thrown into tabular form for ready reading, these numbers appear as follow :—

Total Population 862,346 at different ages.

Under 15 years of age	330,246
15 to 20 ,, ,, ...	100,212
20 to 25 ,, ,, ...	83,563
25 to 30 ,, ,, ...	54,243
30 to 35 ,, ,, ...	44,397
Above 35 ,, ,, ...	249,681

Analysing the numbers more minutely, 107,758 between 10 and 15 years gave only

12 deaths from phthisis, while 183,775 between 15 and 25 gave 253 deaths to that cause. Of the 253 deaths 209 were natives, 181 of them Victorians, with 28 Tasmanians, boasted to be free from phthisis! Even at the more advanced ages 25 to 35, of a total 276 deaths from phthisis at those ages, 101 were of Australians.

Confining remark to Australians, in round numbers 108,000 persons living between 10 and 15 years old gave 10 deaths to phthisis, while 184,000 between 15 and 25 years gave 209, showing a great rise just above puberty.

The increase of deaths from phthisis between 15 and 25 from 157 to 209 in one year, and then onwards to 299 in the year following, as shown on page 25, is a rate far in excess of the numbers growing into the ages prone to the malady.

Few Victorians have phthisis between 10 and 15 years of age; but at the critical turn in young life a sudden rise comes in deaths from the disease How is this? Is the cause physiological or sociological? A medical or a political problem? It cannot be climatic,

for the like happens in other climes? Statists record the fact without note or comment. They are not supposed to deal with matters medical, and wisely refrain. Medical writers generally associate the proclivity to phthisis at puberty to the evolution of the sexual system, but the bacterial etiology of the disease affords a more physiological explanation.

Between the ages of 10 and 15 years there is in all countries a very small fatality from all causes. Disease and death seem then to be at rest everywhere. However greatly they may vary at other ages, at these they are uniform. Only for convenience do our statists divide their life terms into lustrums; but if a finer physiological basis were taken, it would undoubtedly be found that in the second of life's seven stages there is least sorrow. From 7 to 14 bodily ailment is at a minimum. In the first stage the frame goes through violent physiological adjustments, amongst which are obliteration of sundry rudimentary organs of unknown use in the economy, unless they be to ally

the human form to other forms of animal life. In the second stage those changes are completed preparatory to entering on the third stage. This stage puberty begins, and with it all its concomitant trials, amongst which is a proneness to phthisis. With the evolutions going on, the respiratory organs undergo great changes, the chief alteration being marked by the breaking voice. This peculiarity is not owing merely to local modifications of the vocal organs in the larynx, for a corresponding enlargement occurs in the respiratory power to supply air as the mechanical force required for the raucous voice. The new force is gained by enlarged air vesicles and widened openings into them from the ultimate branches of the air tubes. Hence it happens that, when this change occurs, should the individual be allowed to breathe in an atmosphere laden with the germs of phthisis, the poison particles will freely enter the air vesicles, from which before the widening they would have been excluded.

It is not that the germ itself could not

enter an air chamber, for a bacterium is well nigh impalpable and barely within sight of the utmost powers of the microscope, and able therefore to enter wherever air enters; but the vehicle on which the germ is air-borne, a dried pus corpuscle or an epithelial scale, is a body too big to go through the unrelaxed opening into the air-sac, guarded as that portal is by the active ciliated columnar epithelium and acutely sensitive and quickly reflex contracting muscular coat of the ultimate bronchiole.

As the whole theory of bacterial infection of the epithelium of the air-sac in phthisis depends upon the accuracy of the inference about its mechanism, it might perhaps be well to mention that where the air-tube ends in the air-sac the muscular coat is extremely sensitive to irritants and all kinds of stimuli, it having been seen, under experiment, to contract to an almost entire obliteration of the caliber, which is naturally about the 1-50th of an inch in diameter.

It is usually thought the acid dyspepsia so often marking the onset of phthisis in young

people is probably the initial disorder in the series of morbid actions. Very far more probably the early anorexia is only the earliest observable effect of the paralysing power over the central ganglia of organic life of the inhaled fungaceous organisms forming the septic agents in the disease. It is conveyed through the great sympathetic nerve, and especially along the great pneumogastric branches leading direct from the lungs to the gastric ganglion. These micro-fungi have the same acro-narcotic poisonous property as the macro-fungi. By acting in multiplying myriads on the nerve centres regulating appetite, ingestion, digestion, assimilation, nutrition, excretion, the functions of trophic life, these septic micro-fungi will readily depress the vital power, leaving the impaired energy, low temperature and vitality, with listless apathy, forming so prominent features in incipient phthisis. In the midst of these disorders of mere organic life, the organs of the mind and of animal life being left intact, seem to grow brighter by the contrast. Even the partial paralysis of the orbicular

muscle of the eyelids half uncovering the cornea, and giving a marked feature to the physiognomy of incipient phthisis, is another effect of the same acro-narcotic influence.

Not, then, by any vaguely-defined impaired health, referred to generally as predisposing; but alone by an anatomical alteration in the frame at puberty, with co-ordinately altered physiological functions, are young adults made so markedly prone to phthisis. This change forms the only "*vulnerability*," to use a favourite word brought into the medical rhetoric of phthisis to resound, but really explain nothing.

The so-called diathesis is merely bodily debility in feeble frames, the issue of phthisical parentage. The hereditary forms of phthisis are seen in children, who are ever most exposed to inhaling or ingesting the specific virus, amidst which they live and hourly breathe, and therefore oftener fall the readier victims, with a miracle when they escape.

This exposition of a common fallacy about hereditary influence, which was first offered

by me some years ago, would now appear to gain in general acceptance. For example, the *Lancet*, 15th July, 1882, observes:—

> While the discovery of Koch raises into even greater importance than before the inherited predisposition, it will probably lead to some modification of our views as to the influence of that predisposition. Cases may be due to infection which are now regarded as the result of inheritance only. A consumptive mother, for instance, may infect a child through a pocket-handkerchief.

This agreement with my views is gratifying, and shows that when once the clue is got we soon follow up every turn in the labyrinth.

Theory thus vindicating itself as the very backbone of true practice, one might now survey the future prospects of this community in its relation to phthisis, and estimate the economic value of accurate, intimate, pathological knowledge of the malady, without which, indeed, there can be nothing beyond blind groping in empirical tentative effort, and loose talk about prevention. Such a review is the more needful since phthisis, unlike our occasional typhoid fever, never occurs in such outbreaks as create panics which quickly

subside and are soon forgotten, but works away perennially, quietly, stealthily, exciting no deeper emotion than pity for individual suffering, or aversion from objects of trouble and no little inconvenience.

And yet so great is the apathy that, for example, at the recent Social Science Congress held in Melbourne, the word phthisis was never heard at the Medical Section, no more than if *savants* never had heard a whisper about the fell disease that in Victoria tops the list! The cause of the disease has never once occupied the attention of the Central Board of Health; nor of the Medical Society of Victoria, save alone when that body thought fit to challenge the accuracy of the writer's statistics.

Economists have not determined the money value to the State of every unit in a population. That diseases mainly incident to young people trained in skilled handicrafts interrupt the material well-being of a young colony, daily becomes more evident; and a single instance of the kind may do more to impress upon the commercial mind the

industrial bearing of a medical question, than theoretic expositions of the mode in which pathology steps in to explain some relations between capital and labour.

It might be asked—Does indoor labour necessarily involve any morbid condition peculiar to a handicraft? Phthisis is notoriously the disease *par excellence* of indoor life, and to it the question most applies.

Pathology having no polity, the whole tenor of the argument maintained throughout these pages is to demonstrate that not the trade or occupation, but the faulty way in which they are too frequently carried on, does harm, in this climate exactly as in every climate under the sun. Hence, the natural accident of the climate being semi-tropical cannot make the colony unsuitable for indoor trades. As well might we contend that, because domestic service, as proved by the records of the Melbourne Hospital, yields as large a proportion to the aggregate phthisical death-rate here as in England, therefore is this climate unfit for that calling. In the dearth of labourers, our political economists are

continually calling for more immigrants. But a truer economy would make them be more careful of those they already have.

Or perhaps our *patriæ* were politic to hide ugly truths that might hinder comers to enrich the soil with their ashes. Wishing all to look attractive, politicians would not let facts come out. But would it not be wiser and braver, and more befitting fearless State physicians to look steadfastly into the ghastly eye and on the hectic cheek, and pining hopeful young sorrow, and try to find out, and if possible overcome, the cause of them? Even if the cause had been traced by wild speculation rather than, as it was, by calm science, that itself would have been worthier work than trying to evade the difficulty through ironical jibes.

In asking facility for research into the cause of phthisis there was no sort of importuning, but only for leave to use a few statistics then lying unused. With all her lavish outlay on art, science, learning, Victoria gave not even countenance towards carrying on an inquiry into the cause or extent of the fell

disease that cripples her own native-born work-people in yearly-growing greater number. But the mystery has been cleared away without her aid. Meanly repelled by her great Statesmen, I had recourse, as a last resource, to other equally ample and authentic records. There the facts and figures were bought, and in hard cash paid for, that, having been sifted, assorted, and each item placed in its proper site till the synthesis gave to the whole fabric an organic form, soon enabled the proof of the proposition about the negative effect of climate on phthisis to be brought out so clearly that all the later labours that our Statists do are only able to corroborate and confirm. "This was sometime a paradox, but now "the time gives it proof." The *Victorian Year-Book* for 1881-82 verifies the induction of the work *On Phthisis*, 1870, and so was laid the basis for the now elaborately perfect vital statistics of phthisis in Victoria.

Therefore, notwithstanding the denials to a worker who would not be deterred by menace nor diverted by ridicule, the ætiology of

phthisis is at length a resolved problem in the natural history of human disease.

And by whom has the problem been solved? The solution is admitted to have been "*indicated*" in my brochure of 1876. The reader will see that the words then used by me to describe the parasite are the same as are now used by Dr. Koch in his demonstration. Therefore, to me the perception of the destructive parasite must have been then a clear conception by inductive demonstration. Moreover, I then explained the mode of action of the parasite, beyond which no one yet has gone; if, indeed, we are able to exceed an explanation which is already perfect.

A certain planet was inductively traced to its place in the firmament, in the existence of which all astronomers believed and acted on the belief, long before its presence had been telescopically demonstrated. Indeed, the discoverers of Neptune never saw their new-found planet at all. The disturbances in other planets, caused by it, could be in no way else accounted for, and hence they

inferred that a planet must be there, and its presence as foretold was in due time found in the very spot. The after searchers did not go ranging with their telescopes through space in hope of finding by chance somewhat that was new to the view. With perfected means of research, they turned their sight to the place "*indicated*," and at once saw the object of their search. So the Histologist did not hit upon a bacillus by accident. He searched systematically, improved his instruments and methods until he found the parasite that was known before to exist, and without the active presence of which it was impossible to account for perturbations in health, and changes in morbid anatomical structure. In short, he, Dr. Koch, has done precisely as did the astronomer who, following the inductive demonstrations of Adams and Leverrier, was the first to actually observe the new planet.

Now Adams and Leverrier are accepted by all men as the discoverers of Neptune; as such they are honoured and were rewarded. Between their case and this present one

there is a perfect analogy, and not one whit of difference.

If it be impossible to over-rate the importance of the problems in pathology and practical medicine to which the discovery introduces us now, the practical value was before equally great. That it failed to benefit then, was owing to no failure or defect of its own; it was alone the fault of those who were so wedded to the old pathology that they could not think of anything as the cause of consumption save alone the hereditary diathesis, and impaired nutrition.

Wherefore, I fairly claim to have solved the problem of the parasitic cause of tubercle, as I hope to have already indicated the specific antidote.

The term antidote is here used advisedly. The boroglyceride must be antidotal of phthisis if it destroy, by direct action, the *materies morbi* of the disease. Whether it can kill matured bacteria or not, has not as yet been shown by any experiment. That performed by Dr. Koch proved these tubercular bacteria capable of being cultivated outside the living

body, and after many generations able to retain their infective power as active as were those first taken from the diseased body.* But no experiment has yet been tried to find what antiseptic agent would sterilize a cultivation fluid in the laboratory, against the growth or multiplication of tubercle bacteria. The only knowledge which we have on the point is that hitherto alone derived from clinical work. This, taken by itself, is to me conclusive; but it has as yet not been tried by any other observer, as far as I am aware. That the boroglyceride quickly destroys the *materies morbi* of ringworm, in all the stages of that skin affection, mild or inveterate, recent or of old standing, has been by clinical experiment amply shown beyond doubt. By analogy it should, and it does, so act on the *materies morbi* of tubercle. If it kill not the

---

* The term *bacteria* is here used where Dr. Koch employs the term *bacilli*. It is quite immaterial which is chosen. Both terms mean the same objects. The word *bacterion* is from the Greek, while *bacillus* is from the Latin; both signifying a little rod—a rod-shaped parasite. I prefer the Greek word for euphony.

mature parasite, it blights the spores or micrococci that grow into bacteria. It cannot kill the blowfly, but it can kill the blow. These being dead, and the source of local irritation removed, nature is left free to her *vis medicatrix*, which speedily restores healthy healing action. The spores are not set in the texture of the lungs, beyond the reach of inhalations, as argued by an obsolete pathology. They multiply within the air sacs, and these are in direct open traffic with the outer air, and therefore within easy reach of inhaled antiseptics. The parasites are, moreover, buried in the midst of a loose detritus of dead cells, ever undergoing necrobiotic changes and decay. Thus the spray of the boroglyceride solution becomes, as it condenses within the air tubes, practically a local wash to the internal sores, exactly as it is used as a wash to the ringworm sores on the outer skin. There is nothing occult in the behaviour of the antiseptic in disease at all. What is true of the lotion on the surface of the body holds true of it on the mucous surface of the air membrane.

Therefore, we need not defer our verdict on the efficiency of the treatment by this antidote until it can be, by experiments conducted in the laboratory, decided whether or not the boroglyceride can kill bacteria. In such a test one element must always be absent, the vital action of the living body. We cannot possibly tell whether the action of living protoplasm under its nerve influence of life, will correspond with the chemical action of a lifeless test infusion. Therefore, I repeat, the clinical trial is the only valid one ; and when we find it by direct proof, and also by analogy, leading to the one result, there can remain only the one conclusion of an andidotal property, and a true cure for phthisis.

APPENDIX.

THE following is a reprint from the 1876 brochure, in which I enounced the bacterial etiology of phthisis. I omit only references and explanations. The excerpts form an abridged tractate; but the extracts are all consecutive, and are verbally unaltered from the original. They will supply a felt want —the significance of bacilli in tubercle. They give the true difference between morbid anatomy and pathology, the finding of certain results of diseased action, and the thoughts on the nature of the process that lead us to understand in what way the results come about. With a view to explain the parasitic genesis of tubercle, the remarks are now, as they were originally, offered.

THE PATHOGENY OF TUBERCLE.

Tubercle, in the natural disease of the human body, forms in the epithelium on the walls of the alveoli of the air vesicles, the ultimate and true respiratory membrane. This epithelial layer is formed from an exudation of protoplasm from the capillaries of the bronchial arteries, the proper nutrient blood-vessels of

the lungs, the capillary vessels of the pulmonary artery being alone for the respiratory function, and not nutritive of the lung parenchyma. The transudation of protoplasm is, in healthy action, continuous, forming new epithelial cells as the old ones are shed off on finishing their special work.

When, under the morbid influence of the cause of tubercular disease, two or more epithelial cells, with their nuclei, fuse together, they form the giant cell now known to be peculiar to tubercle, a giant cell being only a mass of the *débris* of epithelial cells matted together on the alveolar wall. Lying imbedded in this mass are vast numbers of minute granular bodies of faintly-defined homogeneous structure, the exact nature of which is not yet finally made out. The bodies are, however, invariably found among the giant cells; there can be no doubt about their presence, because they have always been seen by everyone who has looked for them. It is only the significance of their being that is not yet seen by the mental eye; and if they be not microzymes, their precise nature still remains unknown

How, then, would specific micrococci operate as the proximate cause of the equally specific morbid process of general tuberculosis? The *rôle* played by them in the pathogeny of tubercle has never, as far as I can learn, been thus considered; while indeed most pathologists have hitherto done no more than discuss the mere fact of the existence of those organisms, and mainly as incidents among the products of decay. I therefore venture to think they will probably be found to operate as the exciting, efficient, or better termed proximate cause—the *materies morbi* of tubercle, somewhat in the following manner:—

The protoplasm out of which an epithelial cell forms on the wall of an alveolus is chemically composed of a

protein substance holding salts of lime and other mineral ingredients in solution, in combination with phosphorus.

As it is a known action of microzymes, in their mode of life as fungoid organisms, to absorb nitrogen compounds, their proper nutriment, from any albuminoid matter in a changing or an unformed state, such as protoplasm, that they may come in contact with—to feed upon it, and in their way multiply, like other fungal parasites—so will these microzymes naturally operate destructively on the protoplasm of the nascent epithelial cell, withdraw nitrogen, and, with that element inhering kinetic force, liberate pent-up potential energy, and leave the other chemical elements remaining in the impoverished protoplasm, to obey passively their physical statics, and separate by a spontaneous lysis, into an insoluble compound of lime salts with fatty matter, setting free the constituent glycerine, and retaining calcium phosphate in the mechanical mixture. This is the actually known chemical composition of the aggregation of blighted epithelial cells impacted by a process of gradual accumulation, in mass in a lung air vesicle, and termed, according to its mechanical figure as presented to the naked eye, a miliary nodule or tubercle.

In this mass of epithelial *débris* the minute bodies of mycrozymes are set embalmed, or buried as it were in the ruins they produced. To rightly understand the entire process of tuberculosis, it is necessary thus to consider, not only the operation going on in a single air vesicle, but even in a solitary epithelial scale; for of an infinite number of such single actions the whole process is an aggregate.

The same chemical nature enables us to explain subsequent changes that occur, after this necrosis of the epithelial protoplasmic mass, under fatty degeneration,

and further retrograde metamorphosis occurring during chemical decompositions prior to absorption of tubercle, its calcification, or cretification or softening, in the various stages or forms of tubercular phthisis; and it also fully accounts for the peculiar chemical characteristic qualities of tubercular sputa.

The presence of glycerine in the sputum explains the sweetish taste that clincially augurs so ill; the fact also affords an adequate reason why, although sweet, phthisical sputa yield no evidence to tests for sugar; the tests for sugar and glycerine not being the same.

The action thus set up by micrococci would constitute tuberculosis a true mycosis. This febrile state consists in an infiltration into any part of micrococci, with consecutive morbid actions, in the form of local acute or subacute inflammation.

If the explanation here offered be found true, it will fully account for the febrile symptoms occurring on every fresh swarming or multiple of the parasites; also for the consecutive anatomical lesions. In short, this would be found the initiative, and therefore the efficient, cause of tubercle; which, in its turn, is by many admitted to be the irritant setting up the secondary changes that constitute true phthisis. I say *true* phthisis in constradistinction to several forms of chronic wasting disease commonly called phthisis, from their clinical resemblance to the real affection, but which are to be both etiologically and pathologically distinguished.

Eventually the above theory might become directly clinically instructive by pointing towards a rational method of preventing phthisis, and of making it in turn become a "pathological rarity." Or, if not that, of attacking it before permanent tissue change or decay occurred, to secure its arrest, or cure, by agents, such as quinine, experimentally known to be destructive—

germicidal—of the organisms occasioning this epithelial blight. For, while it is but an idle truism to affirm that the healing art never can restore lost normal lung structure, so does it seem premature to assert the improbability of ever finding means of killing parasitic particles in living tissue, without at the same time destroying its own integrity; and this simply because the individual speck of bioplasm forming a *moneron* may have less inherent vital resistance than the formed tissue of the higher organism. Preparations of mercury, iodine, arsenic, and other medicines of the class, so well known for their power of destroying parasitic life upon the exterior bodily surfaces, may operate in precisely the same manner on analogous organisms existing upon the interior surfaces. Finally, from the still later discovery of like organic bodies as essentially causative elements in the histology of leprosy there is derived another collateral probability that the new theory is true. Thus indeed may be explained the effect of mercury in syphilis, and of the famed Tanjore pill, formed of arsenic, in leprosy.

The idea of micrococci being in any way associated with the process of tuberculosis, is a recent one; and the explanation of their mode of operation is, at least as far as I am aware, now for the first time in the history of pathology attempted, with what degree of success remains to he seen.

The view confirms the theory advanced by Dr. William Budd, that allied the affection causatively to the acute specific fevers, and would therefore prove it to be, like its congeners, etiologically independent of the influence of climate.

Professor Küss, of Strasburg, states that "*Tubercle* is "*hypertrophy* followed by a sort of *mummified deposit*, "formed by the epithelium," a definition of tubercle that

agrees with the remark of Frey about "the peculiar "withering of cells in tuberculisation." Those authors, however, offer no other explanation of the mummifying process; any more than do Klebs or Green, at least as far as I have been able to learn, afford an explanation of the manner in which the organisms they speak of operate. A histo-chemical consideration can alone explain the relationship of these organisms to the deposits of mummified or blighted epithelium, forming grey granulations or miliary tubercles, and the further relation of these to the associated phthisical state. It is warmly debated by pathologists, whether the local inflammation or the tubercles come first; but, by the explanation now offered, their occurrence would be simultaneous.

If miliary tuberculosis "has clinically the character of "an 'infective' or so-called zymotic disease," wherein resides the propagating agent? If the fever be like other zymotics, depending on living organisms as the exciting cause, it must obey in that particular the law of the class it belongs to. The enlargement from interstitial growth of the liver and spleen, so common in specific fevers and in tuberculosis, show by analogy that they are probably allied, and therefore alike caused by microzymic contagion. It may even yet be found that in this pathology is contained the key to the explanation of the fact of tubercular disease supplanting intermittent fevers in districts where paludal malaria are removed by drainage or cultivation. This appears a better explanation than that offered of changes in the habits of the people. The introduction of trades and factories into a wilderness means the crowding with population, and consequent increased facilities for propagating the germs of a specific contagion.

By the advocates of the contagiousness of tubercle the germs are usually said to be spread about by the

drying-up of the sputa, the resulting impalpable dust flying up in the air around the sick person; but it has never before been explained in what these germs or particles consisted.

Nor have the very latest researches on the etiology of phthisis apparently advanced beyond the general term. Equally indefinitely does Rindfleisch allude to the giant cells of tubercle as "mysterious objects;" and also "assume that in the primary inflammatory focus a "special tubercular poison is elaborated" in the catarrhal secretions of a scrofulous person, but without further speculating upon its probable nature or genesis. So special a poison must surely be a noticeable entity.

Of the primary nidus of the contagium particles, various views may be held. They may be developed in the morbidly acid alvine secretions, so frequent in tuberculosis, and a condition always favourable for the growth of parasitic organisms. Thus would be associated many of the premonitory intestinal states prodromal of an attack of tuberculosis. The organisms may be the micrococci of Cohn; the microzymes of Béchamp. There is no apparent reason why the microzymes of tubercle should not be as specific as those of yeast or of syphilis.

It is not improbable that a migration of such fungal organisms goes on through the lacteals from the alvine track to the remoter seats of tubercular action. So likewise would thus be explained the "vulnerability" in those hereditarily predisposed, who are only in greater risk through more intimate exposure to the specific agent. The existence of the parasites in single monads, and never in chains or zooglœa, may have added to the difficulty of determining their true nature.

By making various animals—guinea pigs, rabbits, or calves—ingest this diseased milk, Gerlach produced well-marked tuberculosis.

This milk has lately become suspected of being one of the commonest modes of propagating tubercular disease, especially amongst children. When the *milk has been* filtered to deprive it of its solid particles, the fluid portion appears to be as active as the unfiltered. In what form does the virus exist in the serum? If as micrococci, they would not be filtered out of it by any common filtering process sufficient to stop the passage of ordinary milk globules, for those parasitic creatures are less than the 20,000th of an inch in diameter, minor milk globules being perfect giants beside them.

The explanation of these facts would appear to be as follows:—

The secretion of milk goes on through a continuous moulting of the epithelial cells, every cell being but a vehicle for carrying away a globule or particle of milk. If micrococci were present, they would attack the protoplasm of the nascent milk globule, appropriate its nitrogen, and, destroying the vital integrity of the particle, leave the remaining ingredients to form a watery fluid, poor in nitrogen, and apparently abounding in calcareous or earthy matter. This fluid would not only be innutritious, but it would be further actually poisonous when so charged with living contagium of specific disease.

In speaking of the causes of tuberculisation, it is not enough to allude to them as mere forms of impaired nutrition, for pathologists ought to be ready to explain how defects of nutrition lead to specific disease. What is required is a descriptive account of the mode in which the action of decay proceeds. Mere starvation will not induce tubercle. Neither would repletion alone ward tubercle off The use of alcohol often will prevent tubercle, because alcohol, like quinine, kills micrococci. This indeed might be found to be the true rationale of the action of antiseptic inhalations in phthisis. It would

also explain how the same antiseptic property of quinine becomes directly curative of phthisis, by arresting the parasitic cause of the destruction of epithelium. The integrity of the body has to be conserved against the myriads of organisms preying to its destruction. Drugs may be found to destroy the parasitic cause of the disease, as drugs have been found effective in analogous cases: in the power of mercury over the parasite of syphilis, of arsenic over that of leprosy, empirically discovered before being scientifically explained; but in every form of tubercular decay a physiologically indicated form of nutriment must be employed to repair, as far as is compatible with lost or permanently altered tissue, the effects produced by the epithelium invading parasite.

FINIS.

STILLWELL AND CO., PRINTERS, MELBOURNE.

Accession no. ACK

Author Thomson, W.
Germ Origin of Tubercle

Call no. 19th Century Hist.
RC319
V6T36
1882